DENTAL CARIES

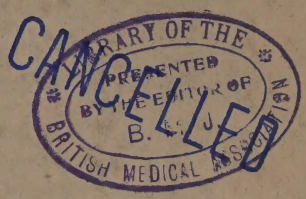
IN RELATION TO ORAL OSMOSIS

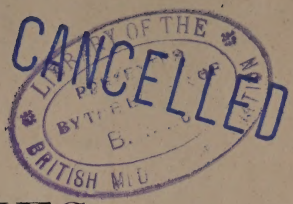
BY

RAGNAR ECKERMANN

ECKERMANN

DENTAL CARIES IN RELATION TO ORAL OSMOSIS



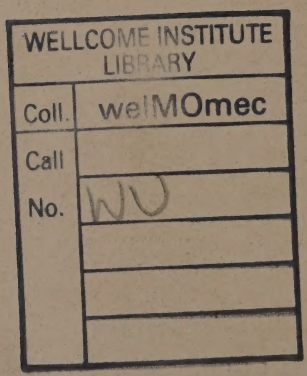


DENTAL CARIES IN RELATION TO ORAL OSMOSIS

BY

RAGNAR ECKERMANN
PH. D.; L. D. S.

LONDON
WILLIAMS AND NORGATE
14 HENRIETTA STREET, COVENT GARDEN, W.C. 2
1919



LUND 1919
CARL BLOMS BOKTRYCKERI

PREFACE.

The main substance of the present work was published at the Congress for Natural Sciences in Vienna 1913 and the same year in Svensk Tandläkare Tidskrift 4. 1913.

On account of difficulties connected with the war it was impossible to get the work printed in England. The reader is therefore asked kindly to overlook a few printing errors that despite the greatest care have crept in as a consequence of the work being printed in Sweden.

Malmoe, Sweden April 1919.

Ragnar Eckermann.

CONTENTS.

CHAPTER VI.

CHAPTER VII.

CHAPTER VIII.

CHAPTER IX.

CHAPTER X.

CHAPTER XI.

CHAPTER XII.

Introduction.

I. Historical Dates.

The classical investigators, from HIPPOCRATES and the writers of the Pharaonic papyrus rolls *(»Papyrus Ebers»)* onwards, regarded caries dentium as a disease originating from »the stagnation of the unsound liquids of the tooth» and discoursed upon »the gnawing of the blood in the teeth» [1]. This explanation — »teoria umorale» (BRUGNATELLI [2]) — was in the course of time transformed into an inflammation theory — »teoria vitale» — which held sway for a couple of centuries and then broke up into theories of »solely parasites» and »solely acids» as the active agents, until, finally, MILLER [3] combined these two opinions into one, according to which the acids in question are produced by parasites — »teoria chimico-parassitaria».

The most important dates in the history of the ætiology of dental caries — mainly according to MILLER (1889), BLACK (1908) and BRUGNATELLI (1913) — are:

a) Authors regarding stagnation of the pulp-liquid as cause: e. g. HIPPOCRATES (456 B. C.), KRÄUTERMANN (1732), BOURDET (1757), BELL (1787), SERRE (1788), KAPPIS (1794);

b) Authors regarding lack of dental nourishment as cause:

[1] The Dental Art in Ancient Times (Souvenir of the sixth Internat. Dental Congress) 1914 p. 11.

[2] BRUGNATELLI, 1913 p. 59.

[3] MILLER, 1889.

e. g. Galeno (131 A. D.), v. Amida (550) and Serapion (1002).

c) Authors regarding inflammation in the tooth as cause: e. g. Eustachius (1574), Hunter (1778), Kappis (1794), Fox (1806), Bell (1831), Abbot (1879), Heitzmann and Boedecker (1886), cf. also Witzel (1880).

It is interesting to read Abbot, who prepared cuts of carious tissue, decalcifying and staining them with carmine. He thus obtained strongly expanded tubuli filled with *yellow* stained »protoplasm» the intertubulary substance being transformed into »protoplasm» in the form of »polykernelled» layers and irregular »medullar elements» [1].

Brugnatelli separates »teoria chimico-vitale» (chemical inflammation) and reckons under this head Tomes (1847) and Colemann (1883), cf. Black [2].

d) Authors regarding parasites as cause: Scribonius (43 A. D.), Ebn-Sina (1036), Kräutermann (1732), Pfaff (1756) and others, who regarded »verms» as parasites. Further: Ficinus (1847), Klenche (1850), Baumgartner (1911) and Fleischmann (1913).

e) Authors regarding chemical substances (org. and inorg. acids) as cause: Lecluse (1754), Pasch (1767), Becker (1808), Rengelmann (1824), Berdmore (1770), Linderer (1837), Robertson (1835), Regnart (1838), Mantegazza (1862), Spence Bate (1864), Magitôt (1867), Wedl (1870), Tomes (1873), Schlenker (1882) and Baume (1882).

f) Authors regarding electrical factors as cause: Bridgmann (1863), Chase (1880), Palmer (1894).

g) Authors regarding sugar as cause: Forest (1597), Pfaff (1756), Ovelgrün (1771), Willman (1860), Colyer (1915).

h) Authors regarding a chemico-parasitic process as cause: Leber and Rottenstein — Über die Karies der Zähne

---

[1] Cited from Miller, 1889 p. 134.

[2] Black, 1908 I p. 63.

(1867); UNDERWOOD and MILLES — Transac. Intern. Med. Congr. (1881); WEIL — Pilze der Zahnkrankheiten, Conferensa a Monaco (1881); MILLER — Mikroorganismen der Mundhöhle (1889).

The different opinions of these authors can be systematized into subjective or constitutional and objective or salivary causes. To the former (caries beginning within the dentine) we then reckon the pulp-plasm and the inflammation theories and to the latter (caries beginning upon the enamel) the chemical, electric, parasitic and chemico-parasitic theories. BRUGNATELLI calculates, as seen, with a »chimico-vitale» theory, which can thus be regarded as forming a transition-stage between the subjective and objective factors.

It is to be remarked, further, that the opinions of the authors mentioned above, are — on account of the complexity of the problems involved — often very vague in the stating of the causes, and several authors have, in fact, opinions which appear wholly conflicting.

II. Criticism of the Theories of Caries.

The modern theories hitherto held of the ætiology of dental caries may be divided into positive causes and negative causes. The positive or objective causes comprize assumed external (salivary) factors, and the negative or subjective ones internal (constitutional) factors.

The Hypothetical Negative Causes are lack of lime (»Subcalcismus») — lack of efficient mastication and cleanliness — lack of congenital or hereditary resistance — lack of potassium-sulphocyanate.

Against the theory of lack of calcium and nourishing salts (»Walkhoff's theory»)[1] the following objections may be urged:

---

[1] WALKHOFF, 1913 cf. with ADLOFF. D. M. f. Z. 3; 6; 12, 1914.

1. It cannot be reasonably asserted that the earth is poorer in calcium salts now than it was formerly, an assumption which would have been necessary in order to understand the great development of caries during the last five decades.

2. Many countries, islands and localities, where the earth consists of nearly pure calcium, are inhabited by people having highly developed caries (cf. Röse [1]).

3. In cases where the chewing is inefficient the saliva deposits a thick layer of dental tartar, which constitutes a certain proof that the human body has calcium salts in surplus.

4. A complete set of permanent teeth weighs about 60 grammes, and it requires but a simple calculation to discover that the approximate annual amount of calcium salts required for the teeth can be obtained from a few pints of milk [2].

Against the theory of inefficient mastication, irregularly placed teeth and lack of oral hygiene (Wallace [3]) the following may be cited:

1. The temporary teeth of young children, who exert only a very weak pressure of the jaws, are frequently quite free from caries.

2. We know many people who exert very little masticatory pressure and who entirely neglect dental hygiene, but who have quite faultless teeth, and, on the other hand, we see people who clean their mouths fanatically, but nevertheless get caries.

3. »Angle claims to have observed a comparative freedom from caries of very irregular teeth.» [4]

4. As a result of researches made by the author with

---

[1] Röse, D. M. f. Z. 1904. 735.
[2] Eckermann, Sv. Tandl. Tidskr. 1908.
[3] Wallace, 1911.
[4] Burchard and Inglis, 1912 p. 332.

a »Gnathodynamometer», invented by himself [1] (s. further Chapter XI) in order to measure the mastication pressure, it was found in about 100 cases that good structure and high pressure went together, but no relationship between caries and mastication pressure was to be seen (cf. below: researches by BLACK and by GASSMANN).

Against the hypothesis of lack of hereditary resistance we can urge:

1. The fact that children as well as their mothers have caries is not at all a proof that it is a question of heredity.

2. The weak jaw with its swiftly erupting teeth resulting in incomplete structure may be inherited, but not the malady. Weak structure may favour but certainly cannot directly cause caries.

»Man spricht von der Vererbung einer Prädisposition zur Caries der Zähne. Diese ist nur insofern möglich, als eine Vererbung von schlechtentwickelten und unregelmässig gestellten Zähnen möglich ist (und auch stattfindet)» MILLER [2].

3. After careful analyses BLACK [3] also rejected the pretention that the congenital structure, »hard» or »soft» teeth, has any influence upon caries. And this BLACK's claim is confirmed by analyses carried out by GASSMANN [4] who found that the molars incl. the third molars have a greater rate of calcium than the canine-teeth.

Lack of potassium-sulphocyanate in the saliva is finally statistically shown as having no direct connection with caries, cf., however, KANTOROWICZ und SPEYER [5] and BRUGNATELLI [6] with LEVY [7].

---

[1] ECKERMANN, D. M. f. Z. 9. 1911.
[2] MILLER, 1889 p. 173.
[3] BLACK, 1908 p. 119.
[4] GASSMANN, Zeitschrift f. Physiol. Chemie 6. 1908. 455.
[5] KANTOROWICZ u. SPEYER, D. M. f. Z. 2. 1914. 81.
[6] BRUGNATELLI, 1914 p. 136.
[7] LEVY, D. M. f. Z. 6. 1914. 436.

»In una recente pubblicazione poi, lo stesso Autore (GIES) si diachara apertamente contrario all'uso interno del solfocianuro, che non solo sarebbe destituito da qualsiasi efficacia contro la carie etc» (BRUGNATELLI [1]).

The Positive Causes advanced for the genesis of dental caries are microorganisms, permanent acidity (local acidose) from organic acids, strong alkalescency, degenerate saliva and the causes embodied in the »sugar theory» [2].

The orthodox theory of the genesis of caries dentium is founded on the chemico-parasitic theory of MILLER and his successors. This theory was advanced in the infancy of bacteriology when there was a strong temptation to explain all pathological phenomena as being of microorganic origin. The great number of other hypotheses afterwards formulated show, however, that doubt has arisen as to the validity of this theory.

The presence of microbes in the carious tissues is a fact, but this circumstance does not prove that the initial carious attack is due to the action of these organisms.

Against the theory that microorganisms cause caries (e. g. BAUMGARTNER [3], FLEISCHMANN [4] a. o.) we may submit the following facts:

1. The animal body protects itself against harmful microorganisms by means of its own — the blood plasm and the phagocytes. Dead teeth, root-filled teeth and gangrenous teeth, which lack living elements, should therefore be more easily attacked by caries than others, whereas the contrary is really the case.

2. In the earth we find skulls containing teeth with caries. If, now, caries is a process analogical with physical death and caused by microorganisms, how may it be explained that these teeth have not become entirely decayed.

---

[1] BRUGNATELLI, 1914, p. 139.
[2] ST. COLYER, Dental Record 1; 12, 1916; 9, 1917.
[3] BAUMGARTNER, D. M. f. Z. 5. 1911.
[4] FLEISCHMANN, Österr. Zeitschr. f. Stomatologie 5. 1913.

The above mentioned arguments show that saprophytic organisms cannot be responsible for the origin of caries.

3. If parasitic organisms of known or unknown type — aerobic or anaerobic — cause caries, strong antiseptics would be necessary when filling cavities in order to hinder fresh caries arising, which is not the case.

4. As an incontestable proof that microbes cannot cause caries, the following case may be adduced: A young lady had gingival caries on two lower molars. The first of these molars also had a proximal cavity. The author prepared this latter cavity and did root filling. The tooth was now dead. The gingival cavity of the same molar was then prepared but not filled. To the cavity of the other molar nothing at all was done. Then half a year was allowed to pass, one year, two years, and now when this is written it is two years and eight months since the tooth was prepared, and yet the prepared, unfilled buccal cavity of the first molar is still unchanged. The bottom may be a little darker in colour, but the walls of dentine are still hard and the rims of the enamel are quite as before. If, now, caries is caused by microorganisms, this cavity, which immediately adjoined a »fresh» one and should itself partly be a result of the work of microorganisms, and which is, further, quite open, would necessarily have been quickly infected and enlarged.

As we know, MILLER himself did not regard organisms as a primary cause of caries upon the enamel, but only as a secondary one. As primary cause he regards acids— organic acids, produced by reversible processes or fermentation of carbo-hydrates by the microorganisms. »Without carbo-hydrates no acids, without acids no caries» (MILLER[1]).

Against the theory that organic acids generate dental caries the following objections may be raised:

1. It is unreasonable to think that just such food as nature has given us in preference to all others, namely,

[1] MILLER, 1889 p. 154.

fruits, roots, bread, milk or their chemical equivalents and which humanity has used since the days of Pithecanthropus, should under non-pathological circumstances be decomposed into components that are able to destroy nature's most resistant animal tissue — the teeth. If, therefore, acidity plays a rôle in the causation of caries, some other substances than carbo-hydrates must leave the acidity [1].

2. If acids could cause caries by destroying the enamel, all teeth having defective enamel of any kind would of necessity be attacked by caries, if caries were at all present in the mouth, because »defective enamel» *eo ipso* constitutes a retention-place.

3. Acids acting alone must cause over the whole tooth or over a great part of the tooth a superficial erosion of indefinite mathematical limits according to the resistance of the enamel, and not deep cavities with small entrances.

4. Among organic acids, lactic acid seems to be regarded by MILLER as the most harmful. If organic acids are able to act upon enamel in the mouth, then, of course, they should more easily be able to destroy dentine and still more easily to dissolve carious matter by destroying the inorganic elements in it. If carious matter (softened dentine) from the same tooth is put into concentrated lactic, nitric, sulphuric and hydrocloric acids, it will be found that, after about 10—15 hours, HCl has completely dissolved the matter, HNO_3 only partly, and that after four months the stuff in the other acids is still undissolved. As a matter of fact diluted lactic acid alone does very little harm to living teeth, although *in vitro* there is produced a superficial erosion on dental enamel free from saliva. Tests with alkaline liquids give — also microscopically [2] — the same result (cf. BRUGNATELLI [3]). Microbes producing lactic acid die in a conc. of 1 + 10000 (MICHAELIS [4]).

---

[1] cf. ÄYRÄPÄÄ, Svensk Tandl. Tidskr. 5. 1912.

[2] BAUMGARTNER, D. M. f. Z. 5, 1911. 350.

[3] cf. BRUGNATELLI, 1913 p. 253.

[4] MICHAELIS, 1914 p. 112.

5. The best evidence of the little value of the organic acid theory is, perhaps, the appearance of the hypothesis which seeks the cause, not in acids, but in quite the opposite direction — the alkalies [1]. But for just the same reasons (points 2, 3 & 4 above) as those for which we rejected the theory concerning acids we are justified in leaving the theory of alkalies. The human body tolerates rather sour liquids, but too strong alkaline ones it tries immediately to neutralize or render weak.

Acidity is under no conditions an independent factor, but only a special quality of the *saliva*, and consequently, it is in the degenerate saliva *ceteris paribus* we must seek the truth [2]. Treating this opinion critically we must ask ourselves what this »degeneration» of saliva in reality implies. If originating from food debris we should certainly be able to protect ourselves against this »degeneration» by careful sanitation, especially in the evening. But as we all know, dental sanitation is quite insufficient as phrophylaxis. Consequently we are obliged to assume that the saliva may at certain intervals contain material harmful to the teeth, which materials have nothing to do with »impurities». What substances do the salivary glands secrete? As a matter of fact we know that the same quantity of chemical salts that enters the body is secreted by the glands of the body and therefore partly by the saliva as well.

What relationship has caries to these products (chemical salts as well as sugar) the enormous consumption of which is due to the development of civilization? And what special significance have the gases, chlorine, carbonic acid and also the metallic gases? Of these the saliva quickly

---

[1] v. Arkövy, Österr. Ung. Viertel. Jahrschr. f. Zahnheilk. 1908 and Preiswerk, D. M. f. Z. 1902.

[2] cf. Black, I 1908 and Lahmann, ref. Äyräpää Sv. Tandl. T. 1912.

absorbs all, which, in addition to the chemical salts present, must increase the osmotic strength of saliva.

And, finally, what is the relationship between saliva and hydrochloric acid, which, coming partly from the gastric juice, partly being a product of catalysis in the mouth, can be detected by careful analyses. This acid is chemically and osmotically very powerful and as a catalyst it is 100—130 times more effective than any organic acid. Whilst the lactic acid needs 3—6 weeks, HCl only needs 3 minutes to annihilate the cuticula (cf. BRUGNATELLI) [1].

As regards the quite new »Sugar Theory» by STANLEY COLYER [2] according to which sugar and easily soluble carbohydrates can generate caries by a »capillary action upon the teeth», we may say that this theory has a good empirical support, as it is an old experience that sugar causes caries. COLYER's theory is in reality based upon the carbo-hydrate theory of MILLER with the SIM WALLACE variation: easily soluble vegetables, with a special leaning towards sugar.

COLYER thinks that in narrow passages, especially at surfaces of contact, it is the »capillary power» that holds the solution sufficiently long to be active. In these »capillary spaces» (pits, fissures and points of contact) a fermentation thus takes place, which explains the arising of dental caries.

A closer study of this hypothesis will soon show the absolute untenability of these claims and that for the following reasons:

1. If this theory were true, wedge-shaped, or spreadly standing teeth, would never have proximal caries, because no capillary action could take place there (between them).

2. Teeth, viz. molars having broad and plain surfaces of contact, which is often the case, must according to this theory have a circular beginning of caries, with a central spot free from caries, but this never occurs.

3. As »civilized» peoples always eat »easily soluble

---

[1] BRUGNATELLI, 1913 p. 253.

[2] ST. COLYER, Dental Record, 1916—17.

carbo-hydrates» and have »capillary spaces», their teeth, if closely set, would always show caries, which is not the case.

4. That in proximate angles, fissures and pits, physical processes in the form of capillary action can take place is probable but that the soluble carbo-hydrates in any decomposed form, even if retained there for a time, should be able to enter the teeth is not at all proven by COLYER.

5. Finally, against this theory the same objections can be raised as against MILLER's of which COLYER's hypothesis is not an improved variation.

In a little paper by WILLMAN[1] we find thoughts concerning the ætiology of caries, valuable to remember.

As causation of caries he regards: Fermention caused by impurities, predisposition, evaporation of chemical substances in certain technical branches, sugar, medicine, especially such with strong mineral acids, hot drinks, crowding teeth, trauma and presence of artifical teeth. He further regards the microorganisms in the carious caverne as a consequense, not as a cause of caries.

The ancient theory of caries being a »stagnation of the bad liquids of the tooth» is not at all to be regarded as a curiosity. It was the general opinion we may say from the days of HIPPOCRATES up to the nineteenth Century. Indeed, the ancient Egyptians seem to have an analogous opinion. As we shall see, ancient wisdom here embodied the truth even if the real cause of the »stagnation of the bad liquids» was unknown. In order to conform to the doctrines of modern medical science the classical conception was in the course of time transformed into an inflammation theory. This theory has also been very roughly treated by criticism (s. MILLER)[2]. Of »calor, rubor, tumor and dolor» we do not see in caries more than rubor and dolor, but this »rubor» is an impor-

[1] WILLMAN, Ystad 1860.

[2] MILLER, 1889 p. 93.

tant fact. The investigators in this field have taken great pains to ascertain from where the colour of caries comes.

According to the inflammation theory the colour of caries originates from blood.

Resumé.

Summing up the criticism of the theories hitherto framed to explain the origin of caries we may state that none of them contains the whole of the truth, but all of them indicate more or less a part of it.

III. Untenability of the Modern Chemico-parasitic Theory.

The summarised Facts.

The most generally accepted theory of the ætiology of dental caries is, as said, the chemico-parasitic one, built up by MILLER and modified by WALLACE, BLACK, RÖSE. WALKHOFF, PICKERILL, MUMMERY and other investigators. We will here give the reasons why this theory cannot satisfactorily solve the complicated problem of caries.

1. In spite of energetic researches covering many decades nobody has succeeded in isolating one or more microbes to which the destruction of the dental enamel can be positively ascribed (cf. SUNDBERG [1]).

2. PICKERILL [2] has compared the bacteriological conditions of the mouths of fifty European children with carious teeth and fifty Maori children having intact teeth, and has found that quite the same kinds of pathogenic microorganisms were to be seen in the hundred mouths.

3. Bacteriological researches of the dentine-canals in very thin sections of living teeth, comprising the stained

---

[1] SUNDBERG, 1915, p. 72.

[2] PICKERILL, The Sixth International Dental Congress in London 1914.

hard part uniting very small carious cavaties with pulp in the direction of the dentine canals have given negative results as regards the existence of microorganisms.

Although these three points have their value, they constitute — as everybody understands — no overwhelming proof, for it is, of course, conceivable that is has not yet been possible to stain the hypothetical organisms, or possibly they are so little that they have escaped the microscopists.

4. In the history of humanity the bacteriological maladies have arisen suddenly, have spread, have been stationary for a shorter or longer time and have afterwards disappeared, but caries has been a scourge of humanity since the beginning of society.

Some diseases, e. g., Lepra, have certainly kept their position since ancient times, but a few thousand cases of Lepra weigh feather-light against hundreds of millions of decayed mouths.

5. Caries never causes cachexia, fever, or swelling of the lymphoid glands like other infectious maladies, and never passes to neighbouring tissues *(per continuitatem)* in the form of caries as other such maladies caused by specific microbes, which first annihilate their favorite organ and then go over to the neighbouring tissues. Not even metastasis *(per emboliam)* is caused by caries.

6. Carious matter is, as a rule, free from smell in teeth with living pulp. This fact, together with other circumstances, is a proof that carious matter is not a pure product of destruction.

7. The chemico-parasitic process is dependent upon the existence of microscopical retention places. It may be definitely claimed that every tooth has one, ay, many, such retention places. If thus, there is caries in the mouth, i. e. one tooth attacked, every tooth would, if a chemico-parasitic process were the actual cause, be successively decayed, which as a rule is not the case. In other words, caries does not infect.

8. It is a singular fact that whereas the body, buried in the earth, will quickly be completely annihilated, the teeth, on the contrary, offer for many thousand years a resistance against decomposition. Among these teeth some may also be carious.

Death has stopped instead of hastening the decomposition of the teeth, whereas in conformity with the hypothetical chemico-parasitic theory, we should have expected the contrary, independently of the question if parasites or saprophytes are the cause of the annihilition. The destruction of the teeth seems, consequently, to be dependent upon a natural power which is irrespective of the common chemical nivelation-process of animal life.

9. If caries were a process generated by acids and microorganisms living in saliva, it would seem to be possible to produce caries in an artificial way by placing a tooth with suitably bored holes into saliva that has been taken from a mouth devastated by caries, the saliva, often renewed and further acidulated, being kept at a temperature of 36.5—37° C. with the help of a thermostat, carbohydrates being also added.

All attempts made by different investigators to produce artifically *the brown cavern* in the dental tissue have, however, been quite unsuccessful and *only white superficial erosions* are produced in this way. Fats and albuminates have the same effect [1].

10. To nature we ascribe a highly developed power of protecting herself against enemies. Despite this we have formed theories according to which the most resistant animal tissue, the teeth which, buried in the earth, have successfully resisted disintegrative processes for many a thousand years, can be destroyed by a decomposition in the mouth of carbo-hydrates; theories, the *ultima ratio* of which is that porridge and milk is harmful food.

»MICHAELS states that the most active dental caries

---

[1] Cf. however, BRUGNATELLI, 1913 p. 53.

is found in the mouths of hypo-acid individuals, in whom etc. . . . and that caries is least active in the hyperacid individuals . .» [1].

The store of acids which in MILLER's theory is *sina qua non* is mainly ascribed to decomposed carbo-hydrates, primarily or secondarily to milk-sugar. Our vegetables, an almost necessary constituent of human food, used as such from prediluvial time, have thus been quite nonchalantly designated as one of the chief sources of caries. Even primitive reason rises against this thought, the fallacy of which, permitted a certain assumption (s. below), we are able to logically prove.

As is known, some animals get caries and are, consequently, not immune to this disease. Among these animals are horses and cattle. The natural and only food of horses and cattle is carbo-hydrates. But in their domestic state they get, thanks to the kindness of man, a little alteration in the menu, viz., artificial feeding stuffs, *sugar* and *common salt,* both of the latter to an extent which it is impossible to think that they get in their wild state. *Assuming that animal and human caries are qualitatively identical,* which is the common opinion, we can draw from these facts the following absolutely logical conclusions:

a) Either caries has nothing to do with food, condiments or anything which is introduced into the mouth, or,

b) If caries directly or indirectly is ascribable to elements introduced into the mouth, it *must* be derived from the additions given by man and not to the food which the animals exclusively consume in their wild state.

c) Granting that the assumption mentioned is correct, both these conclusions eliminate carbo-hydrates as a cause of caries.

---

[1] s. BURCHARD and INGLIS, 1912 p. 339.

[2] cf., however, BLUNSCHLI, Schweiz. Vierteljahrschr. f. Zahnheilk. 1913. 3, with Chapter VII.

11. The supposition that carbo-hydrates play a decisive rôle in carious attacks is also in direct opposition to the known circumstance that this malady has in the last fifty years increased to an enormous degree, especially among the foremost civilized people, or that race which modern ethnologists call the North-Aryan race, or the North-sea people and their transmarine descendants. That among these people the consumption of carbo-hydrates should be greater and the food qualitatively worse prepared than otherwise and before is surely a bold pretention. On the contrary, caries is spreading in correspondence to the gigantic development of chemistry and to the means of intercourse.

Stanley Colyer [1] has methodically studied the food of certain negro people in South Africa. He found that they lived on carbo-hydrates which where carefully deprived of their fibrous components and always dressed in soft sticky forms. The rate of caries was practically nil.

12. The chemico-parasitic theory also leads us to the height of absurdity, namely, that the primary food of humanity — milk — should be able in a non-pathological state of the mouth to be transformed into a substance hostile to the teeth. Why, inconsistently enough, only cow-milk, even if it has a little different chemical composition from mother's milk, is regarded as harmful is inexplicable. Cow's milk has always acid reaction. That milk when consumed produces a weak acidity proves the fact that the gastric juice in babies has a very low acidity. As is known, milk-sugar as well as other elements of sugar can be transformed by the saliva-enzyme into acid. However, the action of the diastase is dependent on the precence of salts (especially sodium chloride) [2]. The lactic ferments die, further, in a conc. of $1+10000$ acid [3], and consequently this production is only of theoretical value. Miller

[1] Colyer, Dental Record 1916.
[2] Michaelis, 1914 p. 75.
[3] » » p. 112.

tries to evade this troublesome circumstance by the assumption that the acid formed will be swiftly transported away by the saliva [1]. It is also to be noticed that many people, viz. Bulgarians, daily consume sour milk. The formation of acids must thus be ascribed to other factors than the lactic acid.

When teeth or sections of teeth are placed in solutions of organic acids, diluted or concentrated, it will be found that the dental tissue offers enormous resistance to such acids. Thus, in one case, sections were lying for ten months in renewed lactic acid (4 + 1000) without a trace of an effect. The solution was afterwards quite clear and had acid reaction.

Furthermore animal food — albumin, fat — yields on fermentation alkaline fluids. A mixed food would then be rather neutral.

Black's (I 1908) »gelatinous membrane» which is said to protect the formation of acids by microorganisms, is thus, — if existing — decomposed into alkaline products as being of animal origin, and will counteract instead of favour the formation of acid.

13. The pretension that »degeneration» contributes to the occurrence of caries is probably based on the thought that a weakening of the mastication pressure takes place, for any other kind of »degeneration» the dentists at least have not to connect with the patients. (As regard consumption of alcohol and caries s. Chapt. VII).

Pressure and caries do not stand, as we empirically know, in direct relationship to one another, for the strongest jaws often show caries, while the weakest ones are sometimes free. The author's own statistics (s. below) in this theme mathematically confirm this known fact. Yet we think that just the enamel, hard as glass, would offer greater resistance than the dentine (s. Burchard and Inglis [2]). In reality our experience confirms the fact that

[1] Miller, 1889 pp. 17. and 20.

[2] Burchard and Inglis, 1912 p. 335.

caries occurs independently of the structure. (cf. Baumgartner [1]). A weakening of the pressure and an accompanying bad structure will, on the other hand, offer less resistance against hostile agents, if these are once at work.

14. If caries were caused by microorganisms only, this fact should demand a scrupulous antiseptic when filling, e. g. simple cavities. But experience has shown that common drying gives a good result, i. e. prevents caries arising at the rim of the filling. As microbes are able to enter and multiply in very narrow passages and hypothetically (according to the chemico-parasitic theory) exist upon the organic substance of the dentine, it is inconceivable that our crystalline filling-matter, e. g., amalgam, should not permit of such entrance, even when the fillings are regarded as first-class. On the other hand, we can see inferior fillings with imperfect margins of enamel, forming ideal entrances, and these fillings have lasted for years without caries appearing, although other teeth in the same mouth are attacked.

The microorganisms live upon organic and not upon inorganic matters, and microbes are the real cause of the pathological changes in the tooth, according to Miller. How comes it then that the carious dentine contains about 25 % inorganic and 75 % organic matter, when normal dentine contains quite the opposite, 27 % organic and 73 % inorganic. *From where comes the addition of organic matter?*

15. Absolutely unaccountable in the light of the orthodox theory are further such empirical phemomena as

a) That a certain tooth has »chronic« or »latent« caries proximally, but open caries at the neck of the tooth. For if the hypothetical active organisms are present at the root they must of course be able to advance into the already existing carious area of the crown.

---

[1] Baumgartner, D. M. f. Z. 5. 1911. 321.

b) That caries attacks certain groups of teeth *symmetrically* in the mouth, but leaves neighbouring teeth, which are often anatomically weaker, quite intact.

c) That broken-off teeth with deep broken furrows do not show any trace of caries, while the neighbouring teeth, on the contrary, show fresh caries.

d) That deep furrows of broken-off teeth are free from caries, but the same teeth have caries at the neck.

e) That in otherwise completely caries-free mouths both the upper corner teeth are carious,

f) That caries not too advanced shows a mathematical regularity as regards its contour.

16. The consequences of MILLER's theory are that caries is preventable by cleanliness and antiseptics. The experience of several decades shows the opposite, and we know that our most diligent and most frequent patients are fanatical mouth-hygienists. No brush or mouth sanitation will be able to save a mouth fromcari es, only possibly to render the carious process a little slower, which, of course, does not mean that mouth hygiene is superfluous; only that it does not constitute prophylaxis against caries.

KIRK [1] gives utterance to the following:

»We are going to save your teeth by preaching the gospel of oral cleanliness; by ringing the chimes upon the statement that clean teeth will not decay; and thus we propose to prevent decay of the teeth in the future, but I ask you to consider with me frankly and honestly, whether that which we promise can be fulfilled by the means now at our disposal».

17. At the third International Dental Congress in Paris the following resolution was passed (paragraphs 1 and 2):

---

[1] KIRK, New Jersey 1912, Speech, ref. v. BEUST, Arch. f. Zahnheilk. 3. 1913.

»Our present knowledge of dental caries cannot explain the different forms of this malady.

It is assumable that caries comes from the inner part of the tooth and grows outwardly i. e. it is an out-breaking malady».

MATTI ÄYRÄPÄÄ writes:[1] »From the fact that the Lapps and Eskimos eat almost exclusively meat and fish, MILLER finds the reason for their good teeth. He has in this connection found that food made from flour and other carbo-hydrates produces during digestion twenty times more acid than animal food. There are, however, other races of people, viz. some Negro people in Africa and in the Philippine Island, who are pure vegetarians and nevertheless have excellent teeth.

OTTOFY found that the rate of caries amongst a tribe living entirely on vegetable food on the Philippine Islands was 6 per cent, and he is of the opinion that the former circumstance was just due to the pure vegetable diet.»

T. VON BEUST[2], who cites this, adds the following remarks: — »Further comments are here superfluous». — He also writes: — »It is to be claimed that *caries is a malady having its origin in the centre of the tooth»*; and »It is quite wrong to take up prophylaxis as a fight against acids and carbo-hydrates, as is now wrongly done . . . To use the chemico-parasitic theory as a guide is wrong from practical, scientific and pedagogic points of view; from a practical standpoint because nothing will be gained; pedagogical, because patients, in this subject, will be misled; from a scientific standpoint it is wrong, *because the theory is untenable* and because the blind belief hinders further investigations in this departement».

Similar expressions by prominent investigators can be cited in plenty.

---

[1] ÄYRÄPÄÄ, Sv. Tandl. Tidskr. 5. 1912.

[2] v. BEUST, Archiv für Zahnheilkunde, 3. 1913. 7.

Resumé.

It is evident that some of the points given above are not axiomatic as being of such a nature that they are impossible to quite confirm (cf. points 1—4). Their value is, however, relative, *i. e.*, together with other points they will strengthen the evidence.

Furthermore, the criticism of MILLER's theory does not involve a rejection of the chemico-parasitic theory in full. It points out, however, that *such a process cannot be the initial causation of caries*, except in such single cases caused *e. g.* by dentures in *dead* teeth (»artificial caries») and further that the oral acidose, when harmful to the teeth, *can, exceptionally only, be a product of fermentation of carbo-hydrates.*

CHAPTER I.

Some Main Features of Dental Physiology in its Relation to Caries.

In order to be able to understand the pathology of the tooth, it is first necessary to know the normal functions of the dental organ, or its physiology. Unfortunately we cannot say that our knowledge of this subject is in any way satisfactory. Our investigators have probed into microscopical and histological problems, but have as a rule shown very little interest for the macroscopical ones.

By physiology of the tooth we clearly mean the normal changes, development, innervation and nourishment of the dental tissues after eruption [1].

The tooth can erupt 1) too early, 2) normally, 3) late, and 4) too late. On account of our knowledge of the embryological (ontogenetical) construction of a tooth we are justified in drawing *a priori* the following conclusions.

The earlier a tooth erupts the greater is the volumetric proportion between weak and hard tissue, between pulp and dentine. The pulp chamber has then its maximum size (physiologically, not embryologically); the walls of the dentine their minimum size, and the protecting membrane has still porosities which even very low magnification will reveal. We also know, empirically, that such small histological depressions are gradually smoothed out and disappear [2].

---

[1] The nourishment and innervation have not been touched upon in this paper.

[2] Investigations by v. EBNER, ref. FLEISCHMANN, Österr. Zeitschrift f. Stomatologie, 5. 1913; cf. Dental Cosmos, 11, 1913. 1123.

These claims, based upon empirical and clinical observations, are fully confirmed by BUNTING and RICKERT: [1] »We checked and combated the statements of H. P. PICKERILL (Prevention of Dental Caries [2]), who said that the teeth, when first erupted, were not dense and well organized as to their enamel surfaces, but that they became condensed after coming in contact with the saliva. We have found that those hard well formed varieties of teeth which are usually associated with high calcium saliva have surfaces which are far more dense and less penetrable to silver salt than are those of an inferior grade. From all of which it seems reasonable to infer that the enamel of the tooth, to a certain extent, is a changeable structure and that when it is first laid down by the enamel-forming organ it is not complete, but that certain spaces exist between the enamel rods for varying distances from the surface toward the interior. Whether or not similar deficiencies or failure of full formation exist in the deeper parts of the enamel and in the dentine as well, we are not certain. But it seems very evident that the tooth normally undergoes a condensation after its eruption.» (cf., however, WALKHOFF [3]).

A tooth erupted at normal time has certainly also porosities on the enamel, but they are narrower, less numerous, and lie farther from the pulp, and would, of course, be subject to shorter exposure to possibly existing harmful agents than in the case of a too early erupted tooth [4].

Late erupted teeth are embryologically more developed and, consequently, have still fewer porosities, if any, in the protecting membrane, and are thus to be regarded nearly as immune to caries, but, no joy without sorrow, for, as known, the risk in the case of a late erupted tooth is that it will not find its correct position. The case is

---

[1] BUNTING and RICKERT, The Journal of Nat. Dent. Assoc. 5. 1918. 519.

[2] s. also: PICKERILL, Dental Record, 8. 1918. 339.

[3] WALKHOFF, 1913.

[4] cf. TÜRKHEIM, D. M. f. Z. 10. 1914. 729.

otherwise with a too late erupted tooth, *viz.*, the wisdom-tooth. Such a tooth often lies for a long time under the mucous membrane, at the same time having a little passage to the mouth, through which passage harmful materials may enter and cause an undiluted fermentation, which in case of bad oral conditions, will cause a destructive spreading process on the fresh enamel. The result is to be seen as a white colouring on the surface as soft and opaque enamel. From this stage »primary» caries can develop itself, but not always. Often, these white superficial erosions of the enamel will undergo a post-eruptive hardening. That we can see especially on the neck of young teeth. The enamel of such a tooth will probably never have reached, even superficially, its maxium hardness, perhaps on account of the absence of the hardening influence of the fresh saliva [1].

With regard to our *embryological* knowledge that a tooth is built up from the periphery to the centre and, further, our *empirical* knowledge that a weak tooth, viz. a »Hutchinson's tooth», has defects of the enamel here and there in the form of rounds erosion and *not* — as could be expected — *general, weak, thin* or *unresistant enamel,* but *local defects,* (cf. Türkheim [2]), we are, however, *a priori* justified in definitely claiming that a too early erupted tooth has a more porous enamel than a normally erupted one.

What, then, is the cause of a too early eruption? the next question will be.

On account of empirical reasons we learn that lack of sufficient pressure at the time of eruption will be the negative cause of teeth appearing too early, and consequently all such factors as deprive the jaws of the possibility of developing its force accelerate the eruption.

Such factors are: —

---

[1] Experiments by Head, Dental Cosmos, 1907. 802.

[2] Türkheim, D. M. f. Z. 10. 1914. 729.

1. A weak structure of the system of bone in general, on account of constitutional maladies or other causes, which abnormally renders the eruption easy and hinders the development of the force of the jaws.

2. Bringing up on the bottle, which as a rule also renders weak the strength of the body in childhood.

3. Caries at an early age, which directly hinders or irritates the function of the pulp.

It is, of course, conceivable that also other, now unknown, factors foster an early eruption or dentition, which conception *inter alia* is possibly based upon the fact that some children are born with teeth erupted.

However, after the eruption of a tooth is completed, the principal physiological function begins: the »retrogressive metamorphosis» of the pulp — the most significant factor of dental physiology. Of course, this function is only a continuation of the embryological one, but the difference is that now (after eruption) this function vill have to fight against enemies which tend to hinder its work. It is evident that this »retrogressive» function is from another point of view — that of the development of the dentine — progressive, and could, therefore, with equal reason be called the progressive metamorphosis of the dentine. It is astonishing to read in some text-books, (s. for instance COHN [1]), that the contraction of the pulp involves *a weakening of the tooth* — not at all a rare conception. MORGONSTERN [2] also disproves this opinion, in illustration of which we cite the following: »Die fortgesetzte Anlagerung neuer Dentinsubstanz im Inneren des Dentingehäuses ist meiner Ansicht nach ein rein physiologisher durch die Funktion bedingter Prozess, der nicht als pathologisch, wie es RÖMER versucht, aufgefasst werden kann» (GUIDO FISCHER [3]). The latter has the opinion that

1 COHN, 1900 p. 257.

2 MORGENSTERN, D. M. f. Z. 10. 1909. 714 cf. with EULER, D. M. f. Z. 12. 1911. 865.

3 G. FISCHER, D. M. f. Z. 6. 1912. 625.

this function of the pulp takes place as a consequence of permanent, strong mastication only and not normally in *all* teeth, as is the case. »Im übrigen aber ruht sie (= the pulp) untätig und geschützt hinter der sicheren Mauer von Zahnbein und Schmelz« (Brasch)[1]. The contraction of the pulp connected with simultaneous formation of dentine is, in fact, nature's way of restoring an organ by concentrically strengthening the inside, which is worn out from the outside but cannot be externally regenerated like other organs. To the retrogressive metamorphosis of the life of the tooth can, in fact, be ascribed the circumstance that the cusp could be ground off, worn out, indeed, the whole crown successively worn away without caries arising.

The mathematical expression of the proportion between pulp and hard tissue, i. e. the state of structural development, can be obtained in the following simple way. Split a young tooth longitudinally, remove the pulp, and clean the two parts with alcohol. Pour, say 1 cm^3 of coloured spirit into a finely marked narrow glass measure. Immerse the sections of tooth suspended in a fine silk thread under the surface of the liquid. Let V_0 denote the result. Now fill up both the half pulp-chambers of the tooth with wax. Immerse tooth again in the measure. The result this time we denote as V_1 and consequently the formula

$$\frac{V_1 - V_0}{V_0}$$

is the mathematical expression of the structural proportions of a tooth.

The figure should never exceed $^1/_{10}$, which is the approximate expression of a too early erupted tooth. The lower the value is, the better. A normally erupted tooth yields a result of about $^1/_{15}$, a too late one about $^1/_{20}$ [2].

---

[1] Brasch, D. M. f. Z. 6. 1911. 421.

[2] cf. Trueb, D. M. f. Z. 6. 1909. 401.

Practically, the retrogressive pulp-metamorphosis indicates that a weak tooth, if it is left untouched by caries, gradually becomes stronger and stronger (quite as much as a tooth normally erupted), which implies that the proportion between organic and inorganic matter in the tooth is augmenting in favour of the latter. This fact, which is not only empirically revealed but also easy to see by splitting carious teeth of different ages, makes the prospects of prophylaxis more promising.

During the time the dentine is growing stronger the enamel is being fully built up by internal deposits. For parts of the tooth where the consolidating process has reached a certain point, the enamel has lost its property of being a protecting membrane. That this state is early reached — one to two years after eruption — for instance, at the cusps, we can see on teeth wich have a broken corner or which show congenital erosions without caries in the cusps, when caries otherwise appears in the mouth, yes, upon the tooth itself. To regard the enamel as the only protecting factor of the tooth (which is the common opinion) is wrong. The new-dentine lying close to the dento-enamel junction has as regard power of resistance against chemical agents the same properties as enamel and we should also be justified in calling it new-enamel. The enamel and the gingiva is, however, the first line of defence, new-dentine, the second one. What new-dentine histologically means we do not know. It is partly known in the text-books under the name of »Zone of transparency» (the same name is unjustifiably given to the dentine uniting the carious area and the pulp). Empirically we can only say that new-dentine is to be regarded as the result of a consolidation process, which terminates in the formation of a product which is more resistant against chemical and physical influence than dentine in young teeth or that the formation of new or post dentine *perhaps* is the transformation of an active living tissue into a neutral one. That the new-dentine to a certain degree lacks circulation and

innervation we know empirically from worn-off teeth with revealed dentine (cf. the root-dentine). MILLER [1] regards the »Ersatz-dentine» as dentine that has diminished its organic components, but not directly obtained any addition of inorganic matter [2]. According to BURCHARD and INGLIS new-dentine (»secondary dentine») is associated with *tubular calcification* [3].

In people who use their jaws energetically a half part or more of the crown is commonly worn away at the end of life. From this it follows — *eo ipso* — that the weakest points of the enamel for the longest portion of life must be in the border-parts at the neck (gingival belt). First at rather an advanced age are these parts quite impermeable to destructive processes.

Thus the superposition of layers of new-dentine begins at the top, as we can see by splitting the teeth, and gradually proceeds in an apical direction. In the case of a forty years' tooth (owner about fifty years) the whole pulp can be found as a thread in the middle of the root, and the crown is quite homogenous. The author's personal opinion, for which objective proofs cannot of course be submitted, is that the formation of new-dentine and the consolidating of the tooth is the only *raison d'être* for the existence of the pulp, and the purpose of the enamel is to protect the depositing process. MORGENSTERN [4] has the same opinion. The common assumption of regarding cracks on the enamel as the origin of caries is probably quite wrong, in any case it is not empirically confirmed, rather the opposite. The same may be said of the opinion that it is harmful to brush the teeth with hard materials, *e. g.*, pulverized pumice. On the contrary, the more mechanical grinding, artificially by brushing, naturally by chewing, the more

---

[1] MILLER, 1889 p. 126.
[2] cf. KANTOROWICS, D. M. f. Z. 8. 1910. 545.
[3] BURCHARD and INGLIS, 1912 p. 427.
[4] MORGENSTERN, D. M. f. Z. 10. 1909. 713.

we use our teeth, provided that they are sound, by cracking nuts, breaking hard biscuits, indeed, we will even dare the paradox that the more mechanical violence the enamel and dentine suffer, the stronger the tooth will become, for the dynamical injury and pressure hasten the retrogressive metamorphosis of the pulp, and the dentine changes into a tissue which can endure the attacks of its enemies. But this consolidating process goes on slowly and — excuse me for once again repeating it — from the cusp to the apex, and consequently the neck of the tooth is the most exposed place on the tooth if the crown itself has escaped caries. Nature tries to counteract this circumstance by *slow* eruption. *The susceptible necks of the teeth together with large parts of the crowns are more or less concealed by gingiva many years after eruption.*

It is now time to consider to what degree the normal physiological process is influenced, *i. e.* temporarily or permanently stopped, by the enemy of the tooth, »caries».

We all know that the enamel forms a cap over the tooth which is longer buccally and lingually and shorter approximally. Caries through the enamel we can call crown-caries, in other cases »neck-caries» or perhaps »root-caries» would be preferable.

Crown-caries is thus a process that passes through the enamel. Its *sine qua non* is a door or entrance through it. We now empirically know that in mouths where caries otherwise appears — the harmful agents thus being at work — teeth with large defects of different kinds on the enamel (large open entrances) are nevertheless free from caries. We can then draw the conclusion *that not only a pure defect of the enamel is necessary* but also that this defect must be of quite a special kind, and in any case not caused by an agent operating externally only, *e. g.*, acids or other caustics. Irrespectively of what is the real cause of caries we further know that between the initial defect and the pulp is a distinct, macroscopically visible, stained, anatomic connection (the »caries canal» or anastomosis).

This assertion is to be regarded as a fact, even if an overlooked one.

We also know that the pulp contracts in the direction of cusp to apex. Consequently the appearance of caries [1] and the contraction of the pulp stand in a certain relationship. What relationship?

On account of studies made of a large range of split carious teeth, together with what is written above, the author feels himself justified in advancing the following thesis: —

1. The contraction of the living pulp diminishes correspondingly to the possibility of crown-caries arising.

2. The possibility of getting crown-caries is consequently limited to a certain early period of the life of the tooth, unless no artificial factors, trauma, operation, dentures come into play.

3. The points attacked by crown-caries are gradually moved from the cusp in the direction of the neck of the tooth.

4. These points cannot be — on account of empirial reasons — pure anatomic defects of the enamel, but must be of a physiological kind, *i., e.,* standing in a certain relationship to the life of the tooth.

5. The enamel is structurally porous (histological pointlike defects), in its early years (months) but is then undergoing a post-eruptive hardening, The »caries-canals» in the crown are consequently laid out at this time.

6. Between caries and pulp (here the protecting weapon of nature) now begins a fight, the result of which may be:

a) The pulp conqueror. The »caries-canal» is early closed up and caries remains as a yellow-brown to brown-black area on the enamel and as a yellow canal in the dentine from the pulp to this zone.

b) Pulp and caries equally strong. The caries

[1] = »Natural» caries: independent of human operations or arrangements.

slowly grows larger in order possibly to reinflate later into open caries.

c) Caries conqueror. The attacks not so seldom coming from different points lead to an early annihilation of the crown.

7. This annihilation is of:

a) A physiological nature: larger and larger parts of the dental tissue gradually being deprived of the protecting influence of the pulp and of life;

b) A pathological nature — microorganisms wandering into the unprotected »canal» and successively infecting the pulp, after the death of which — *ceteris paribus* — the tooth is theoretically condemned.

Irrespective of what the cause of caries may be, the conditions promoting *immunity* [1] to this malady have been found to be: —

1. Immunity to crown-caries:

a) The eruption of teeth in fully built state. A rare case amongst »civilized» people.

b) In the case of teeth having in some way abnormal structure, absolute freedom during and after eruption from the influence of harmful products.

c) The complete healing up of the »caries-canals» which commonly appear in permanent teeth, *viz.*, the canals to the approximal and mastication surfaces (= »chronic caries»), and the retraction of the crown pulp to the region of the root.

2. Immunity to root-caries:

An undamaged (not retracted) condition of the gingiva, and especially the papillae, and the pulp-contraction proceeding in normal order.

3. Immunity to gingival erosion:

---

[1] Concerning the idea of immunity s. »Criticism» f othis chapter.

Gingiva not receded. Low acidity (on account of absence of HCl) and ample consumption of pure common water and the retraction of the root-pulp far advanced.

As a summarized conclusion we can thus say that caries retards, stops, or annihilates, the retrogressive metamorphosis of the pulp and in the same degree the physiological life-process of the tooth. That too-early-made root-fillings are not suitable has been confirmed empirically.

If we ask ourselves, on the other hand, what factors favour this process of the pulp, we shall find that we can divide them into artificial and natural ones.

Artificial:

1) Metal fillings; 2) Grinding of the cusps.

Natural:

1) Wear and tear of the teeth; 2) Hard mastication-pressure.

That metal fillings form a good means of forcing the pulp down to the root we can easily see in metal-filled split teeth. It will be seen how the pulp has built up round the filling a thick wall in spite of the fact that the total contraction itself is not so far advanced. Empirically we know that metal fillings are the most lasting and that they are a means of strengthening the teeth. The relationship between fillings and the pulp-contraction is such that young front-teeth may be filled with metal (Au or amalgam). Crown fillings have, of course, the most »pressing down» effect on the pulp; in the second rank come approximal fillings. A mesial, a distal (and a central) metal-filling together seem to be necessary in order to press the crown-pulp quite down and to ensure no further caries arising, provided open or closed (chronic) caries is at hand.

At the Congress for Natural Sciences, Section Odontology, in Vienna, Sept., 1913, Müller — Vienna, read a paper in which he stated that it was his opinion that the grinding of the cusps of the teeth was a prophylactic means

against caries. The auditors probably smiled inwardly, but he was right, in fact, even if he did not know the real cause of the result he asserted, or rather had experienced, for he said he had used the method with good effect for a long time. In consequence of what is said above, it must be clear that everything which stimulates the retrogressive process is at the same time a prophylactic against caries. That to these stimulating means belongs not only the grinding of the cusps, but all mechanical violence, consequently also wear and tear of the teeth, is evident. From our practice we know that for purposes of good occlusion, when fitting artificial crowns, we can grind off the top of the antagonist without a trace of caries being seen later on. We also daily see people who have worn out their teeth until half of the crown or more has gone, the top of the tooth often forming a deep retention-place, but nevertheless no caries has appeared.

We have now seen how the extreme consequences of our premise — the connection between the very important retrogressive metamorphosis of the pulp and caries (see further Plate I and II) — is confirmed by our long experience as well as by facts daily manifested by nature herself. And as the correctness of the strictly logical consequences is empirically proved, the starting-point must be correct.

As known, a bone of contention among our leading men is whether the enamel is a »*living*» tissue or not [1]. It is to be seen from what is written above that the incomplete structure of the enamel can, if the normal physiological process of the tooth is not interrupted, successively complete itself to normal structure. On the other hand, we know empirically that teeth made pulpless artificially (by cauterization or extirpation) or pathologically

[1] cf. *inter alia:* Rygge, Skand. Tandl. Förs Halvsekeljubil. 1916, with Walkhoff, D. M. f. Z. 1898; 1901. S. further Hredsky, ref. D. M. f. Z. 5. 1915. cf. also: Walkhoff, 1913 with Adloff, D. M. f. Z. 3. 1914. 169; 6. 1914. 454; and 12. 1914. 836; cf., finally. Black, Dental Cosmos 1895 with Röse, D. M. f. Z. 1908.

resist caries in the mouth as long as living teeth, and that »dead» enamel is consequently not to be regarded as a dead product of the body.

»Emaljdannelsen og emaljens forkalkning begynder ved kronens spids og fortsætter nedover kronen efterhvert som denne vokser, saa man paa kronespidsen kan have fuldt udviklet og forkalket emalje paa same tid, som den findes i sin aller förste udvikling paa de dybere liggende dele af kronen; desuden vill den del af emaljen, som ligger nærmest dentinet være det mest udviklede og stærkest forkalkede, medens den del, som ligger nærmest ameloblasterne er den minst forkalkede, solange tanden er under udvikling.» Haus [1].

On account of these and other known facts, we can claim:

1. That during formation (ontogenetically) the enamel is a living tissue.

2. When full grown — a »neutral» tissue (without combustion and renewal).

3. Both these states must have a *transitory state* as regards *time* and *place*.

4. This transitory state as regards *time* is the *normal* eruption-time, or immediately before.

5. This transitory state as regards *place* does not simultaneously comprise the whole coat of enamel, but begins at the cusp and ends at the neck.

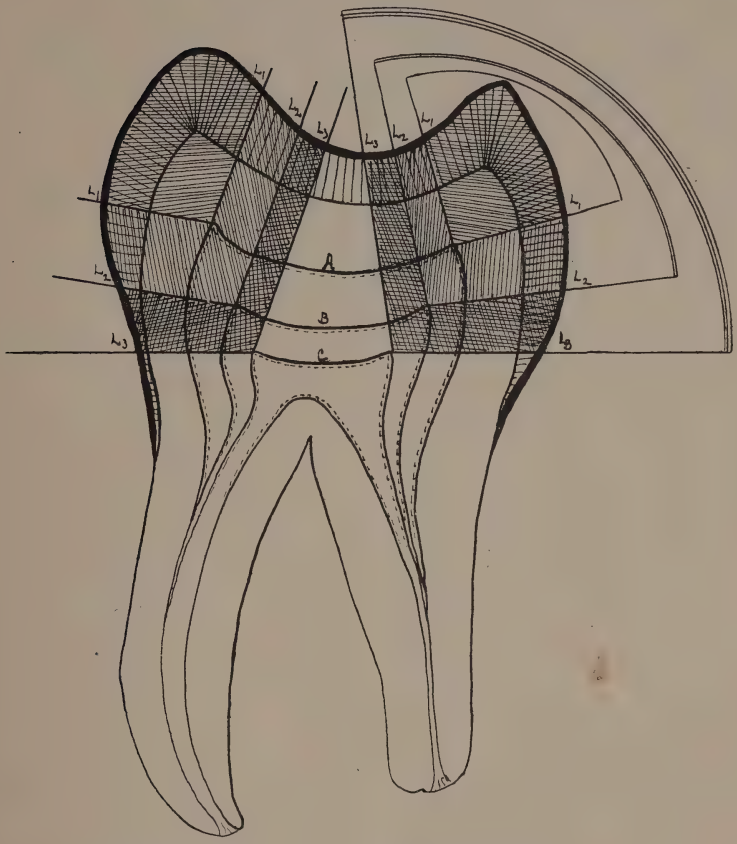
6. A tooth can consequently have its enamel on the top »neutral» and at the »upper» part of the tooth still under development (sedimentation).

---

[1] Haus, 1917 p. 160. »The formation and calcification of the enamel begins at the top of the crown and continues down over the crown in relation to growth. One has then fully developed and calcified enamel on the top of the crown at the same time as it is to be found in its very early stage of formation in the deepest part of the crown; further, that part of the enamel lying close to the dentine may be the best developed and most calcified, whilst that part lying nearest the ameloblasts is least calcified as long as the tooth is in the state of formation»

Plate I.

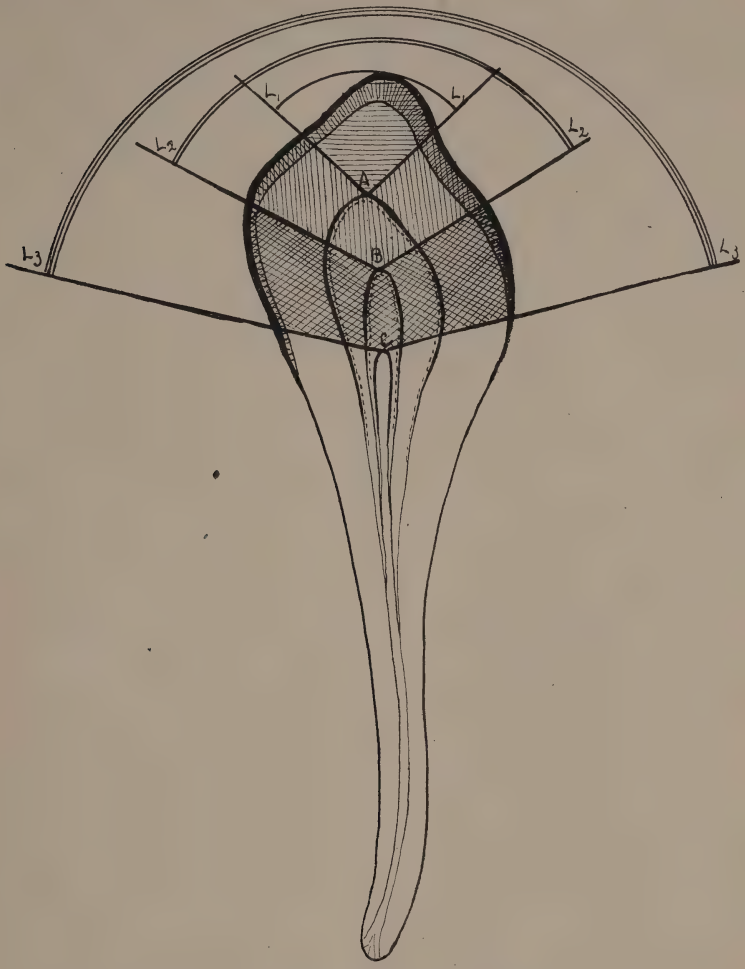
Schematic Diagram of a Molar (Premolar) Type.



L_1--L_1 = Zone of immunity at time of eruption.
L_2—L_2 = » » » about 5 years after eruption.
L_3—L_3 = » » » » 15—20 years after eruption.
A = Roof of the pulp at time of eruption.
B = » » » » about 5 years after eruption.
C = » » » » » 20 » » »

Plate II.

Schematic Diagram of a Tooth of Cuspidal Type.



L_1—L_1 = Zone of immunity at time of eruption.
L_2—L_2 = » » » about 5 years after eruption.
L_3—L_3 = » » » » 15—20 years after eruption.
A = Point of the pulp at time of eruption.
B = » » » » about 5 years after eruption.
C = » » » » » 20 » » »

7. A too early or too swift eruption will expose not fully-built enamel (round the neck).

It is evident that the living parts of the enamel are those which are most exposed and most sensitive to attacks from the enemies of the teeth; also, of course, the revealed parts (the neck) which quite lack enamel.

Summary.

1. A tooth of cuspidal type is more favourably situated with regard to the attack of caries than teeth of molar and premolar types, because the latter ones have — theoretically in any case — a central zone in the mastication surface which is never »immune».

2. The conic tooth presents the ideal type as regards physiological immunity to caries.

3. Two factors further increase the condition of immunity

a) The properties of the new-dentine (»Ersatz-dentine») to render difficult a possible passage.

b) The increased distance between the pulp-chamber and the external wall of the tooth.

4. The diminishing of the pulp varies considerably. In normal cases (*e. g.* no caries and hard mastication) the crown-pulp has disappeared and the crown already becomes quite homogeneous in 10—15 years after eruption. Persistent resorption-teeth (deciduous-teeth) have in the thirtieth year of the bearer no pulp at all, neither in the crown nor in the root.

5. The neck of the tooth is during the whole life a susceptible part, and the good condition of the gingiva and the papilla is a very important factor. On account of slow eruption the crown of the tooth is not quite free from gingiva for many years after eruption.

Criticism.

Immunity is absolute or relative. Absolute immunity does not exist, we may claim, whether in man or in higher mammalia. Relative immunity, advocated here, comprizes either

a) single teeth *in toto,* or,
b) local regions of single teeth.

These rules of immunity concern, of course, only teeth, existing in natural anatomic conditions, and not teeth used as support for artificial arrangement, for instance, bridge-work, denture or anything like it, under the protection of which a strong mineral-acidulated decomposition takes place, which is able to break down the enemal or the external structure of the tooth.

CHAPTER II.

The Caries Canals.

Anatomic Properties.

If sections are made of teeth in which caries is not too far advanced, it will be found, *provided the sections have been made in the right plane*, that a faint, rose-coloured or yellow-coloured streak or zone can be seen stretching from the carious point or area, if ever so little, to the pulp. These streaks follow the dentine tubules, and are always in the direction of the pulp, never in another direction. Good illustrations of these streaks are shown on Plate III, IV and V. The sections can be easily prepared. The tooth may generally be split in a mesial-distal direction and ground with a file or carborundum and then with sand-paper discs until the right level has been found. In case of early caries the zone is very thin and a few strokes of the file too many will obliterate it. At times it does not become clearly visible until the tooth has been thoroughly dried. It is more than remarkable that nobody has taken up the work of establishing the significance of these marks. They are, nevertheless, in the highest degree characteristic and prove macroscopically a clear geometrical connection between the pulp and the carious initial.

It may be pointed out here that these zones never have a gabled or forked appearance. In the literature these canals are partly treated and mentioned under the name of »Zone of Transparency», derived from the superficial fact that very thin sections containing this »canal» look optically

transparent (»hyaline area», according to BLACK [1]). But under this name other phenomena are included, *e. g.*, old dentine lying under the cusps, which is quite another dental formation manifesting the state, when dentine begins to acquire the hardness of enamel, and, further, the dentine of the roots [2]. The »canals» are, further, not considered as an *inseparable* and *unconditionable* factor of caries. Their genetical character is explained as a consequence of »irritation from outside» [3]. This is certainly so, but what kind of irritation is meant? »In cases where the softening and the pigmentation has not yet begun, we obtain a pure image of transparency, in which cases one cannot yet speak about caries of the dentine. Here the transparent part is forming a cone, the point of which is directed towards the pulp» MILLER [4].

The pretention that the carious zone or carious canal should be of an embryological nature is contradicted by the fact that the canal from small carious defects is very thin and becomes broader and broader from growing caverns, and, further, by the circumstance that teeth with exposed roots, after splitting, as a rule show such canals from the neck of the tooth in the direction of the pulp, *but in teeth without exposed roots canals (to the surface of root) are always absent.* It is easy to see that caries canals coming from exposed necks are standing in relationship to the size of the exposed part

---

[1] BLACK I 1908, fig. 117, p. 97, certainly says concerning an illustration showing just a typical caries canal: »The *forked* projection of the flamelike tongue, formed by the *border of cloud* (= caries canal) etc.» In reality his figure is an excellent support of our claims.

[2] »I tynde slibninger blir dentinet gjennemsigtigt, transparant diaphant dentin, som meget almindeligt optradder i roden». (In thin sections the dentine is transparent: diaphanous dentine, which very commonly appears in the root.) HAUS, 1917 p. 138.

[3] s. a. o. CAUSH, Dental Record 2, 1916. cf. with. MILLER, 1889 p. 124: »Die Ursachen der Transparenz».

[4] MILLER, 1889 p. 122. cf. with BLACK, 1908 p. 70.

Plate III.

Caries Canals.
Photo.

of the root. Thus, the caries canals cannot possibly be of a congenital nature but must be acquired.

As already said and as can be seen in the illustrations these streaks run along the tubules the shortest anatomic route to the pulp and have towards the pulp a smaller width, corresponding to the radial course of the tubules. The author has given the name »Caries Canal» to this streak. This channel always has a faint rose-colour. The oral liquid is probably never yellow-red or in any case only sporadically; the pulp fluid is always yellow-red. Does the colour originate from blood plasm?

Blood and Iron Tests.

If an osmotically strong solution were be able to affect the liquid of a living tooth, the process would theoretically have to take place in such a manner that the water of the osmotically weaker solution is streaming to the former. Now the matter in reality stands thus, that the erythrocytes or the red blood corpuscles »obey the laws of the isotonic co-efficients» (HAMBURGER)[1] *i. e.*, blood is very sensitive to osmotic influence. If, thus, the genesis of caries in general is an osmotic process, and if, furthermore, caries is a hemal process, then there should be found traces of blood in the caries canal and in the walls of the carious cavity. To ascertain if this were so, samples were taken of the canal from excised (not extracted) teeth. The samples were taken out of the walls of the carious cavity, though not until the split tooth had been carefully scraped clean, the dried pulp removed, the sides of the pulp cavity bored clean, the upper part of the carious matter taken away and the section of tooth washed several times in benzol. All these precautions were observed in order to preclude the possibility of the blood penetrating from elsewhere. Further, the samples were taken out of carious

[1] HAMBURGER, 1902 I p. 27.

cavities in their early stage in the mouths of living persons (*i. e.*, not from skulls). In these cases such mouths were selected as did not normally bleed and from which no teeth had been extracted for years. From fissure cavities with a very narrow entrance the upper layer of carious dentine was removed, the softened dentine was excavated and for the purpose of examination taken out, washed in benzol, and pulverized in an absolutely clean crucibele.

The analyses were at first made in accordance with Rossel's aloin test, which in all cases yielded a positive result [1]. Like the Guaicum and Benzidine tests and all other chemically unknown colour reactions, the aloin test, which some writers [2], to be sure, consider very reliable and so exact as to show blood in 1 : 25000 dilution, probably has, nevertheless, only a negative value. A negative result excludes the possibility of hemal colouring matter being present. A positive result can, however, be attained by means of other materials [3].

In his text book COHN [4] writes with reference to dental nourishment:

»The following experiment by MILLER speaks for a transudation through the dentine: If crushed carious teeth be treated with hydrochloric acid and then with potassium ferrocyanide, a blue colour appears at the place where the carious and the normal dentine meet, which is a sign that an iron salt is present here. As, according to the author's (COHN) analyses, iron cannot be found in sound dentine, MILLER's assumption must be correct, *viz.*, that the composition of iron in the pulp-blood, after being reduced to

[1] The matter has to be washed in water, the solution acidulated with a few drops of acetic acid, after which the same quantity of ether is added. After shaking 10 drops of hydrogen superoxide 10—20 drops of solution of aloin are added (2 per cent. Barbados or Natal aloin in 90 per cent. alcohol). After some hours a beautiful, sherry-red colour appears.

[2] SCHAER, Zeitschrift f. Analytische Chemie 8. 1903. 42.

[3] NEUBURG, I 1911 p. 937.

[4] COHN, 1900 p. 256, cf. with MILLER, 1889 p. 70.

Plate IV.

Caries Canals.
Photo.

Plate V.

Caries Canals.
Photo.

soluble compounds of ferrous oxide, ooze out through the dentine and are oxidized by oxygen to insoluble compounds of ferric oxide on surface.»

From this it follows that MILLER and COHN have proved the presence of iron in carious dentine, and its absence in sound dentine, which double relationship has been confirmed by the writer in about 100 cases by means of the very delicate potassium-sulphocyanate reaction [1].

Presence of iron may mean presence of blood, absence of iron must mean absence of blood. It follows, therefore, that carious matter may contain blood, sound dental tissue not. The carious tissue contains something else than non-carious tissue and, consequently, it is not a common destructive tissue, but a positive transformed one.

Compare the following analyses:

1. **Analysis of the Enamel:**
(after Berzelius)

88.6 % Calcium-phosphates c. fluor
8.0 % Calcium-carbonide
1.5 % Magnesium-phosphate
2.0 % Organic substances.

2. **Analysis of the Dentine:**
(after Berzelius)

28.0 % Organic substances
64.3 % Calcium-phosphate c. FlCa
5.3 % Calcium-carbonide
1.0 % Mg-phosphate
1.4 % Na, NaCl.

---

[1] Dissolve carious matter in boiling concentrated nitric acid (only two or three drops), add 5 c.c. dist. water, 3 c.c. potassium-sulphocyanate (10 %), and finally 3 c.c. ether. Shake and close up the glass pipe. After 10 minutes the ether will show a rose-red colouration (Iron-rodanide). To be sure that the acid (or some of the other reagents) do not contain iron, it is of course necessary to make check tests of the reagents only. If then the colour of the real test is stronger than that of the check-test, the result is positive.

Analysis of the Salts of the Saliva:
(after JACOBOWITSCH).

Potassium-sulphocyanate	0.07 %
Alcaline chlorids	0.84 %
Phosphates of soda	0.94 %
Ca and Mg	0.04 %
Sulphates of soda	traces

Thus, dentine, enamel and saliva do not normally contain Fe [1].

If we ask ourselves whence the iron comes, it may be pointed out that both MILLER and COHN give the pulp, and indeed the pulp blood as source and exclude all others. It may also be remarked that the observations made by these authors were not of a single tooth, but of a large number of carious teeth.

Instead, however, of the simple explanation at hand that the iron had been transported by the pulp blood MILLER and COHN go astray in the above fantastic explanation, which is inexplicable from all points of view. Why and how can the »iron» by itself leave the erythrocytes in the pulp, pass through the membranes, and the dentine-tubules up to the cavity by changing from ferric to ferrous-salt and from ferrous to ferric again? It is strange to think that during his long years of valuable research on the cause of caries, MILLER was at precisely this moment nearer the truth than earlier or later. The reason that MILLER sought for iron in the carious dentine, he says, was in order to find the source of the specific, permanent colouration of caries. The author's own search for iron was a consequence of the conception of an osmotic process as explanation of the arising of caries and the positive analysis constituted at the same time a confirming fact.

The passage cited from COHN (l. c. p. 256) concerning iron tests made by MILLER and COHN, independently of each other, are, however, misleading, in so far as the

---

[1]) cf. GASSMANN, Zeitschr. f. Physiol. Chemie 6. 1908. 460.

test is reversed by them. They are best carried out in the following way:

If carious teeth, carious matter or sections of teeth with caries be put into 2 per cent. potassium ferrocyanide for, say, 2—4 hours and then taken up with wooden pins and placed into 2—10 per cent. HCl, the most characteristic and absolutely decisive blue (blue-green) [1] iron reaction will be found, commonly after some minutes (or even seconds), but sometimes first, though then more clearly, after some hours. The thinner the section the shorter the time in the acid. After the blue colour has distinctly appeared the matter should be taken up with wooden-pins or glass rods and carefully washed in water, after which it can be seen that the blue colour, *i. e.*, the iron, lies in or originates from the carious matter or from the pulp-walls, and not in the sound dental tissues.

It is obvious that for the successful execution of this test the following conditions are necessary:

1. That the object employed must be so selected or so treated as to exclude the possibility of blood coming from elsewhere;
2. No iron instruments whatever must be used;
3. The acid must be quite free from iron (control test);
4. The tube must be well closed.

The following rather complicated method is, however, more advisable [2].

A weak, but not too weak, carious tooth is taken and carefully cleansed mechanically by grinding and then washed in benzol or alcohol, the caries canal being afterwards ground out by grinding on both sides of the tooth, so as

---

[1]) At first blue-green, and then almost blue. It is to be observed that 12—24 hours after placing object in acid a green post-colouring is setting in almost always, which post-colouring is not to be regarded as a positive result.

[2] cf. Molisch, 1813 pp. 2, 3.

to obtain a very thin section of tooth in which the caries canals are seen as weak striations in the dentine. It is of advantage, if as much as possible of the configuration of the tooth can be kept understroyed, but the walls of the pulp may be bored clean. Then the section should be carefully cleansed once again and placed into 2 per cent. potassium ferrocyanide [1], where it ought to remain 2—4 days in darkness. In the ferrocyanide solution it can remain for any length of time, not so in the acid. After it has been taken up and placed with the help of clean pins of wood into the acid (2—10 per cent. HCl.), it must lie here for some minutes only. Too long a time will destroy the whole section and, further, make it quite blue (or spread the strong colour to the edges of the section). The section should now be taken up and transferred to a glass containing aqua dest., where it is repeatedly and carefully washed in different waters. Now it may be taken up and placed on the object glass, embedded in Canada balsam or examined directly.

If the preparation of the section is not correctly performed the result will be that a blue colour appears at the walls of the pulp and the sides of the root; the test then yields a negative proof. The blue colour manifests the presence of blood. We have in the walls of the pulp and round the root remains of the gingiva and gum, which always contain blood and consequently also iron. A section, hardened in formaline and afterwards decalcified in phloroglucin, would theoretically be still more suitable for this iron-test, as it could afterwards be cut into thinner sections. But this method is too uncertain, because of the risk of getting impurities containing Fe.

A magnification of about $\frac{50}{1}$ to $\frac{100}{1}$ will now enable one to see in dentine tubules belonging to the caries canals

[1] This concentration is given for plants and used by the writer with good result. It is, however, not excluded that for inst. 5 p. c. would give a quicker result.

small blue points lying rather sparely here and there, but not in the sound dental tissue, if it is carefully washed. These points, which often are numerous nearer the pulp wall, the latter being quite blue, proclaim the presence of iron — blood iron, hematin — and at the same time the presence of blood, which has wandered from the pulp to the enamel. Even if some little spot of the section should be superficially stained by the strong colour, it will be easy to see that it is a secondary staining only, not having been taken away by the washing. A light grinding will, of course, remove this stain.

If this test is well executed the section can be photographed on a plate which is especially sensitive to blue, and in that way the section with its blue spots can be demonstrated on a large scale by a projector.

Teichmann's test of hemin crystals (brown, rhombic crystals) was made, as well as the Teischmann—Kobert's test, which differs from the former in so far as NaCl is exchanged for iodide of potassium, the crystals then having an intensive black colour, making them easily discernible under the microscope. With the exception of some cases in which, on account of the small quantity, no definite conclusion could be reached, the tests (fifty) gave a positive result and thus established the presence of blood.

The Teischmann's test of hemin is theoretically very simple, as it only consists of a heating of the matter with NaCl and acetic acid, but in practice this test is rather difficult to accomplish, especially in this case on account of the *indissoluble nature* of the substance under consideration. It is advisable to bore a very little matter from the carious canal with a clean drill, to wash the powder with chloroform or benzol and then to add some grains of pure sodium chloride, and finally to heat, not boil, with a drop of acetic acid. Often it must be heated several times. Strong sunlight is probably the best means of producing crystals, also it takes a much longer time. By magnifying about 2—300 times one is able to see the small dark brown,

transparent, (by moving the mirror) pointed, rhombic crystals, which generally are to be found in the periphery of the subject. It is to be observed that in these tests, other large light-red, rhombic crystals — possibly hemoglobin(?) crystals — and also large, black crystals, which have one obtuse rhombic end (the other end being square), are to be seen, neither of which kinds should be confused with the hemin crystals.

MILLER writes[1]: »The sections of carious dentine stained with picro-carmine often show peculiar images *which are not always easy to explain* . . . . . Otherwise the parts being in decomposition are commonly *yellow*. In the soft but not infected parts as well as in the crowds of fungi the *red stain* is predominant. Lying scattered, strongly expanded tubules are filled with a *bright yellow-stained* homogeneous substance etc.» — If, for comparison, we stain centrifuged blood with the intensive red picro-carmine[2] we will find that just *the dried plasm appears upon the object-glass as a bright yellow-stained product* (cf. ABOTT in the »Introduction» of this work).

Though the result obtained was perfectly sufficient (the Teischmann's test is valid as medico-legal evidence),[3] the author also tried to ascertain the presence of blood by means of spectroscopic examination, but did not succeed in doing so. Although a check test on Teischmann's crystals was made in at least two cases with positive result, the spectroscopic test gave no decided result, only indicating that the matter »in all probability» contained blood.

Tests were made of hemoglobin, hemin, synhematin and lutein. It is probable that the hemochromogen-test would be preferable. (cf. HAMMARSTEN[4]

[1] MILLER, 1889 p. 146.

[2] Dissolvable in alkaline water (some drops H_3N and Aqua).

[3] »The suggestion made by the author (ECKERMANN) that the yellow streaks in dentine are due to the colouration by iron from the hemoglobin of the blood seems a very reasonable supposition». BUNTING, Dental Items of Interest 1918.

[4] HAMMARSTEN, 1889 p. 68.

That the result was partly negative is probably to be ascribed to the insignificant quantity of the material tested, as well as to the difficulty of dissolving carious matter. (Carious dentine is according to the author's researches only soluble in HCl and HNO_3.) Concerning lutein (the colouring matter of plasm) it may be stated that the chemical composition is unknown, and that it may be transmuted by fermentation, or, as in this case, by diffusion. Moreover, lutein is so constituted as to bleach essentially in the presence of other materials or sunlight. The absorption-bands of lutein lie, further, in the blue and violet, where the eye can only with difficulty distinguish differences. As a matter of fact spectroscopic evidence as to colouring matters of blood and their by-products is only reliable in combination with Teischmann's crystal test (or Kobert's or Donogany's hemochromogen crystal test). Teischmann's tests are undoubtedly in themselves absolutely decisive [1].

The spectroscopic investigations were carried out by the University Institute of Physics in Lund. The performance of the blood tests mentioned was intentionally assigned by the author to other persons so that the result should in no way be influenced by selfsuggestion. The investigators did not know the purpose of the research and were consequently impartial. Most of the experiments were checked by the author.

Resumé.

1. In the carious canal or the connection between carious initial and the pulp *Fe is present,* while sound enamel, dentine and ash of saliva contain no iron.

2. In the carious canal of the dentine *blood is present.*

3. These facts prove that the carious canal constitutes a *passage for plasm.*

[1] Neuberg, 1911 I p. 934.

Critiique.

The iron-analyses used here are very reliable and among the most exact ones in inorganic chemistry. With pure reagents the iron-tests are very easy to perform, but the risk and suspicion that iron can be derived from another source is always at hand. This risk is eliminated in the case of the more complicated, but safer, blood test.

Colour of Caries.

As is well known, blood has the quality of coagulation. This happens as soon as it comes into contact with air. Certain chemical salts also have the quality of causing blood to coagulate. The blood plasm coagulates thus from two causes when it reaches the zone of the enamel. Here it calls forth the familiar yellowish-red to reddish-brown colour on the tooth. The tubules are therefore darker here than nearer the pulp-wall. This colour cannot originate from decomposition in the mouth only, for in that case every depression of the teeth and especially deposits on the teeth as well as on the tartar (except due to smoking) would exhibit the colour. That the small black points upon the teeth likewise are derived from blood is very probable and it can be confirmed that mouths containing such have a bleeding gingiva. Roots cut off up to sound dentine and covered with dentures also do not show this reddish-brown colour (provided it has not already appeared in consequence of the cutting-off and grinding having revealed root-caries). The reddish or dark-red appearance of caries is thus partly derived from plasm — in fact from pulp plasm.

In some cases the colour of the canal is but slightly perceptible *e, g.*, in the bottom of a prepared cavity, probably on account of the rapidly proceeding destruction, which does not always allow the plasm to become fully coagulated before it is dissolved and transported away by microorganisms. However, an extracted, split and *dried*

Osmotic Tests (schematic figures).

Fig. 1.

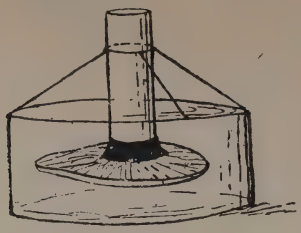


Fig. 2.

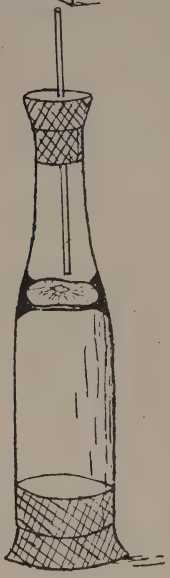


Fig. 4.

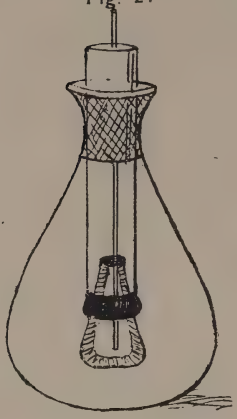


Fig. 5.

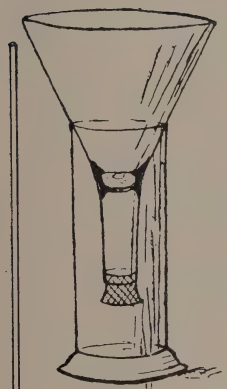


Fig. 3.

Fig. 6.

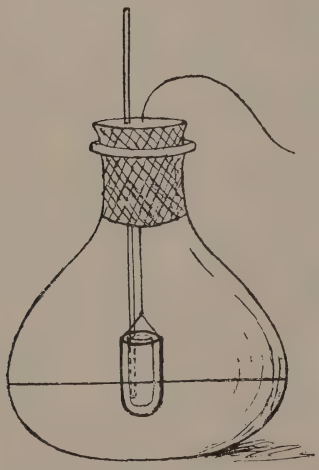


Fig. 7.

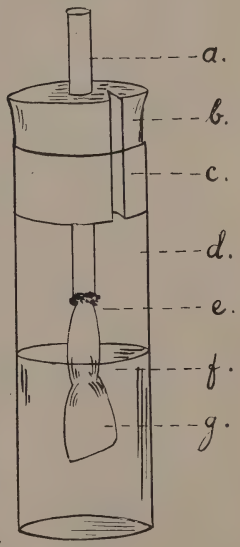


Fig. 8.

Figs. 1--6. Tests with sections of tooth.

Fig. 7. Check-test with a tube of silicate cement.

Fig. 8. Test according to CAUSH a) Small glass tube passing through cork. b) Cork. c) Slit in cork to equalize pressure owing to any alteration of temperature. d) Large glass tube. e) Cement for attaching tooth to small glass tube. f) Fluid. g) Tooth.

The fine pipes in Figs. 3—7 indicate shortened osmometers.

tooth always shows the characteristic connections with the pulp. The swifter the carious process the lighter is the colour, says also MILLER, among others. Chronic caries is the darkest. The explanation of this fact is simple; the coagulation requires a certain time, if not left without disturbance it will not have time to coagulate fully.

Osmotic Properties of Dental Tissues.

For the experiments involved, sections of teeth, longitudinally and transversely cut teeth, and whole teeth with extensively bored-up pulp-cavity were used. These were fixed in glass tubes in the manner indicated on Plate VI. For insulating and fastening purposes the author used, after several unsuccessful attempts, cement and amalgam in single or double layers, as well as gutta-percha, covered with glass-wax [1]. The sections were about 1—2 mm. thick. As solutions the following were used: — Aqua dest, physiological solution of common salt, concentrated and diluted solutions of common salt, Ringer's solutions, lunar caustic solution, as well as solutions of cane sugar, saltpetre and iron salt of different strengths. As stains, carmine, methylene blue and copper sulphate were used. Verification tests were made both before and after each experiment. Those made before consisted of examinations into the absolute density of the insulation. For this purpose a weak solution of lunar caustic was used. If only a fractional part of a drop penetrates the insulation into the NaCl-solution, the solution produces an instantaneous reaction, an opal-coloured precipitate being formed. (Water from the main contains chlorine.)

The check tests made subsequent to the experiments were intended to show whether the colouring was really diffuse or only superficial. These consisted of grinding,

[1] »Picein» is perhaps better, s. WALTER, Ann. d. Phys. (4) 18. 1905. 860 (ref. SVEDBERG, 1912 p. 102); BUNTING uses Canada-balsam (The Journal of Nat. Dent. Ass. 5. 1918. 521).

after the test, the section of the teeth concerned, as well as simply placing other teeth and sections in the colouring solution in question to determine whether the colour could capillarily or only osmotically penetrate the dentine. The dental tissue of the root apices is very porous and consequently not suited to these researches. In cases where no stains, colour-solutions or *osmometer* were used it was ascertained by reaction with solution of lunar caustic that common salt (in any case the chlorine) had diosmosed through the teeth. The diffusion of sugar was proved by means of Almén's sugar-reagent.

The result of these osmotic tests, which were executed in the summer of 1913, and, as should be emphasized, concern dental tissue *in vitro* and comprised about forty experiments, was as follows.

The sound enamel is almost always impermeable or only slightly permeable to salts.[1] Only in isolated cases, where the enamel exhibited the well-known white spots, did it prove sensitive to osmosis. The sulphate of copper and methylene blue show a strong tendency to enter into the enamel from the inside, but not to force their way through. It is difficult when using stains in this experiment to decide exactly, if staining or osmosis has taken place. Common dentine, on the other hand, is almost without exception permeable to common salt, sugar, saltpetre, lunar caustic, water as well as dyes, although only in the direction of the dentine tubules, and this easier in the direction from the pulp to enamel than the contrary. Only new-dentine as well as the dentine of well-formed old resorption teeth (deciduous teeth), or of very old teeth in general, seems to be impermeable or only slightly permeable. The dentine seems only slightly permeable to sugar and saltpetre, whereas it seems always to be impermeable to iron salts.

The positive results of osmosis generally manifest

---

[1] cf. Bunting, The Journal of Nat. Dent. Ass. 5. 1918. 51 (s. below).

themselves very quickly, in a few cases after some hours, at most after twenty-four to forty-eight hours.

The result of the author's osmotic investigations, for the first time published at the Congress of Natural Sciences in Vienna Sept. 1913[1] are, finally, in the most decisive way confirmed by the investigations of BUNTING and RICKERT[2], which after very exact and lasting experiments, came to the following conclusion (P. 526): »From the evidence, which we have gained, we are convinced that the enamel and the dentine of the teeth are as a rule more or less porous and that they will admit of a passage of salts and solvent both from the blood to the surface and from the saliva to the interior, and that such flow takes place in a variable and fluctuating manner due to change in the composition of the blood and saliva.»

According to these investigations enamel *in vitro* is permeable (without limitations?) and this question will be discussed later on[3].

It should be added that CAUSH[4] has carried out some investigations of the osmotic properties of the tooth. The method of investigation with coloured hypotonic in the tooth and unstained hypertonic liquid around it is, however, not so good. The result of his experiments were: — »Though some of the teeth used in these experiments were slightly decayed, there was in no case any sign of colour in the fluid at the end of the experiments, the grinding down of the teeth showed no stain either in the enamel or the dentine, *except at those points of the enamel where the decay had commenced, and here the stain was very slight*» p. 338). From this we can draw the following conclusions: The enamel is in the test impermeable, the new-dentine also, except where it is decayed.

MANNING[5] writes: »the dentine is semi-permeable

[1] ECKERMANN, Svensk Tandl. Tidskr. 4. 1913.
[2] BUNTING, The Journal of Nat. Dent. Ass. 5. 1918. 519.
[3] cf. ECKERMANN, Svensk Tandl. Tidskr. 5. 1918. 309.
[4] CAUSH, Dental Record 5. 1915. 337. s. also Plate VI fig. 8.
[5] MANNING, Dental Cosmos, 1. 1919. 21.

under conditions favorable to the development of osmotic pressure».

Röse writes [1]: »If exchange of matters in general takes place in the enamel of fullgrown teeth it can only be within narrow limits *by osmosis* with help of the dentine».

That the dentine in general must permit of osmosis is evident from the test of Bigelow and Bartell [2] who, by a successive condensation of coarsely granular sedimentation of a precipitate of barium sulphate, continually arrived at membranes which in an increasing degree allowed of osmosis. Already at a width of the pores in round figures, 0.7—0.6 μ, the osmotic work was beginning. It should be remembered that the dentine tubuli have a width of 2—4 μ [Cohn]; 1.3—2.5 μ [Miller], *i. e.*, 2—7 times wider.

Histological Researches.

Many facts speak in favour of the conception that carious matter and the carious pulp-canal may contain not only blood matter, but also undestroyed erythrocytes. If red corpuscles could be detected in the carious dentine, we should have a very easy demonstration and at the same time a strongly convincing proof of the claim of hemal action being a factor in the arising of caries. Amongst these facts the following may be cited: —

1. The red corpuscles are very sensitive to osmotic influence.

2. The red corpuscles measure normally about 8 μ and the dentine tubules only 1.3—2—4 μ, but the erythrocytes are able to shrink under osmotic influence and are in general very elastic [3].

3. The red corpuscles actually diffuse through animal

---

[1] Röse, D. M. f. Z. 1. 1908. 3.
[2] ref. Höber, 1911, p. 190.
[3] cf. Hamburger, I 1902 p. 184.

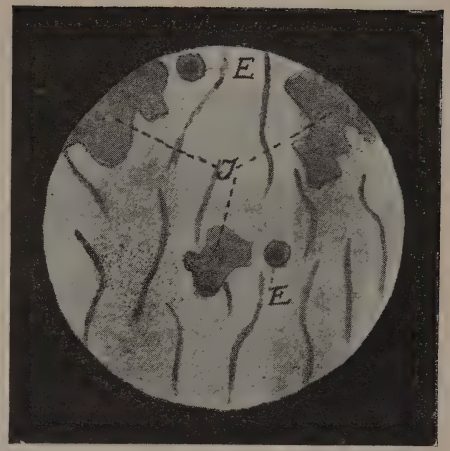
Plate VII.

a

Cut of tooth. 1/28 mm. Magnif. 700 : 1.

K = Carious Cavity.
E = Erythrocytes.
I = Interglobular Spaces.

b



Cut of tooth. 1/28 mm. Magnif. 700 : 1.

E = Erythrocytes.
I = Interglobular Spaces.

membranes, *i. e.*, they wander under certain conditions through the walls of the blood vessels into neighbouring tissues.

4. They change under osmotic influence their biconcave form into a round one.

5. The transudation of blood matter through dentine is empirically known. In the tubules of such a tooth (with transuded blood) as was extracted and prepared, Docent FLEISCHMANN, Vienna, and a collaborator found long rows of undestroyed erythrocytes [1].

6. The osmotic canal of a tooth of normal appearance, the approximal surface of which had a cavity of extra small depth, was ground on both sides and the section thus formed was cut out.

This section parallel to the dentine tubules and about 2 mm^2. in size was, after considerable work, ground down to 1/28 mm., and then handed over to an assistant for microscopic examination. By magnifying 100/1 the carious anastomosis can be seen as striations of a dark colouration running parallel to the dentine tubules. The result gained by magnifying 700/1 is in part visible on Plate VI, where hypothetically three red blood corpuscles are to be seen immediately within the carious wall. One of the corpuscles is situated in an interglobular space. The preparation was kept two days in Ringer's solution, and the cells have in consequence possibly swollen. In all, six blood corpuscles are probably visible in the section.

The author was not personally in a position to check the microscopic examination of the two sections illustrated, and, in consideration of the deceptive construction of the dentine tissue, takes up for the present a tentative attitude towards the possibility of undestroyed erythrocytes being present in the carious tubules.

A section of a tooth was stained in hematoxylin-van Gieson's dye. Although there were several suspicious yellow

[1] Personal communication, Vienna, Sept. 1913.

cell-like formations to be seen, the author does not venture to draw any definite conclusions.

7. Further, an histological series of transverse sections of split carious teeth was prepared; but because of lack of time hardening in formalin was not effected. The sections were at first decalcified in phloroglucin and acid nitr. for three weeks and then cut into leaves of 35 μ — this being, on account of the hardness of the material, the minimum possible with the microtome used — and stained with Hansen's hematoxylin-eosin. The sections were examined under the microscope, the result being negative, probably because of the fact that they had not been hardened first.

This investigation was once again repeated. As stains, hematoxylin-eosin, hematoxylin-van Gieson and hematoxylin-Biondin were used. (These stains recommended by Prof. SJÖVALL, University of Lund, were probably not suitable to the tests in question.) This examination yielded a negative result.

8. From the researches of RÖMER[1], FISCHER[2], BRASCH[3] and others we know that the pulp of intact teeth is subject to atrophic changes. Especially the last-named (BRASCH) describes a series of investigated cases which form a very instructive and valuable addition to the subject. He writes concerning his histological researches: The pulp looks »rather flat and shrunk»; the odontoblasts are subject to »a tension, which appears to tear apart the tissue» . . . The observations made of the cells in the walls of the vessels showed »nucleal indigence up to hyaline degeneration . . . . The tunica externa forms »no close encompassing girdle, but spreads itself thread-like in the surrounding tissue, so that all borders between the two are obliterated»[4].

These conditions constitute a plain manifestation of

[1] RÖMER, Atlas der patolog. Zahnpulpa 1909.
[2] FISCHER, D. M. f. Z. 2. 1910 and D. M. f. Z. 8. 1912.
[3] BRASCH, D. M. f. Z. 6. 1912.
[4] BRASCH, D. M. f. Z. 6. 1912. 411.

the influence of a force which acts from the pulp outwards, and which has evidently deprived the vessels of their fluid and corpuscles. Brasch (l. c.) also speaks about a formation of gases in the pulp.

None of the above quoted writers can give any plausible explanation of these phenomena, only purely indistinct conceptions being advanced. Brasch (l. c.) regards the cause of this atrophy to be lying in the pulp itself: a physiological atrophic inactivity or a too early death. Fischer has the opinion that we have the result of »regressive disturbances» of nourishment [1]. He speaks of a »differentiation of the odontoblast» and seems to have the opinion that the mystic atrophy is caused by chronic inflammation with œdema, which opinion Brasch regards himself to have proved to be wrong (l. c.). Römer speaks about »senile atrophy», which can take place relatively early. (cf. also Walkhoff [2]).

Brasch certainly says that his investigations embraced »faultless» teeth. It may be safely asserted, however, that the common, small brown, superficial zones (»primary caries») upon the enamel were not taken into consideration as being »caries». These small, yellowish-brown areas, which are often hardly perceptible (especially those in the fissures and on the proximal sides) *and which are found practically in all permanent teeth shortly after eruption*, are identical with the earliest stage of caries and the external sign of the secret anastomosis with the pulp chamber. The averment of an osmotic influence upon the pulp, which influence is constantly depriving the latter of its fluid, and possibly also has an influence upon its red corpuscles, gives us a satisfactory explanation of these atrophic changes in the living pulp.

---

[1] Fischer, D. M. f. Z. 8. 1912. 624.

[2] Walkhoff, Mikrophotogr. Atlas 1897.

Resumé.

1. There is no doubt that the pulp-plasm is sensitive to osmotic influence and plenty of reasons are given for this opinion in this work.

2. The erythrocytes are in general also osmotically very sensitive.

3. That the erythrocytes of the pulp can be influenced by osmotic powers effecting the tooth has been made very probable.

Criticism.

If it were possible to demonstrate caries canals containing erythrocytes it would constitute an overwhelming proof of the existence of a power affecting the blood of the pulp from the outside. But *exactly* how far the osmotic influence in ordinary cases of caries in different states of development disturbs the sensitive red corpuscles of the pulp-cavum, is still escaping our knowledge.

Summary of Chapter II.

1. There is not only an anatomic connection (viz., the tubuli) between the pulp and the carious initial, but also a physiologico-pathological one.

2. In carious teeth the dentine tubules running from the carious point to the pulp have a very distinct appearance, being weakly rose or yellow-coloured. In this work these diseased tubules are called *caries canals.*

3. The characteristic colour of the caries canals is due to coagulated blood plasm.

4. *In vitro,* tests show that enamel and new-dentine do not always allow osmosis, young dentine does so. That the dentine thus allows diffusion can theoretically and experimentally be regarded as proven. The existence of the physiologico-pathological caries canal formed by plasm obtains its physical explanation in that way.

Criticism.

The caries canal here described is due to the natural or common caries. One can also, as will be seen in the following, calculate with an artificial caries, arisen in mouths where dentures or artificial arrangements of different kinds are applied to or in the adjacent neighbourhood of *dead* teeth and which through the presence of metals cause strongly mineral-acidulated fermentation.

The difficulty of establishing the real source of the colour of caries becomes apparent, when it is remembered that plenty of materials are able to give to the dentine and enamel the same staining, viz., tobacco, cocoa, coffce etc. dyes from the food, further *blood,* coming from bleeding gingiva on account of traumatic causes, brushing etc.

CHAPTER III.

Attempts to Produce Artificial Caries.

As is well known, no investigator of caries has succeeded in artificially producing that characteristic phenomenon which we call caries, *viz.*, the *brown-black cavern* in the dental tissue — nor even the dark colour only. *Superficial white erosions* are all that the investigators under the name of artificial caries have succeeded in producing.

If caries really was, as MILLER's creed maintains, a pure product of fermentation, *viz.*, microbes and acids from carbo-hydrates, we should most certainly be able, by strictly following certain measures of precaution — thermostat and repeated renewal of fresh saliva from a mouth in which caries is highly developed — to produce at least a shadow of the carious process or in any case to be able to develop caries areas (»primary caries») into caverns. *All such attempts, however,* have been, made, in vain [1]. Caries is, therefore, apparently a process of life, a process of the blood; and the essence of life, the pulp plasm, cannot artificially be produced *in vitro* from itself and this circumstance is a negative proof that caries cannot be of a microbic or fermentation origin *only*.

A. In Vitro.

Against the possibility of producing caries artificially (according to osmotic points of view) outside the mouth, the following facts may be urged.

---

[1] The more or less primitive trials of MILLER (1889), MAGITÔT (1867) and others with corroded teeth stained with tobacco,

1. That the ductility and other qualities which the red blood corpuscles exhibit under the influence of life are to a great extent limited outside the body. Of course, it is not at all certain that the erythrocytes take an active part in the earlier stage of caries, the pulp plasm alone possibly sharing in this.

2. That the dentine tubules of a prepared section have but little chance of possessing the qualities favourable to diffusion of organic liquids.

3. That for practical reasons diluted blood must be used, which makes the formation of the colouration characteristic of caries impossible or but slightly perceptible.

To counteract the fibrination we can add 1.5 per cent. sodium oxalate solution (0.1 gm. to 100 c. c. blood) or Hirudin. Theoretically the blood of the musk-deer, the red blood corpuscles of which measure 2.1 μ. (Bois-Reymond [1]), would perhaps be especially suitable for this test.

My experiments were carried out in room temperature with horse and calf blood, mixed firstly with Ringer's solution, and secondly, with hypertonic salt solution (= hypertonic to blood, say 2 %). The insulating and fastening arrangements were the same as those used for the investigations of osmosis (chapter II). The fluid in which the samples of blood were immersed consisted, firstly, of a hypertonic solution (of NaCl) and secondly, of a hypotonic solution (aqua).

The following tests were made: —

The sections of teeth were placed in tubes each containing five drops of blood and 2 c. c. Ringer's solution, and these tubes were immersed in 10 % NaCl-solution.

After three weeks two of the sections showed

---

cocoa etc. can hardly be regarded as »caries dentium», cf., however, Brugnatelli, 1913 p. 53.

[1] Bois-Reymond, 1913 p. 13.

exceedingly faint, hardly perceptible, yellow stains, the other sections none at all.

With an electric centrifugal casting apparatus horse blood was centrifuged and in each of the five tubes were dropped five drops of this fluid, now being poor in erythrocytes, and after adding 3 per cent. NaCl solution, the tubes were immersed in aqua.

After eight weeks one of the sections of teeth showed a central area with a strong, yellowish-red colouration, two others exhibited faint, yellowish-red tints, and the others had suffered only a slight change. Some of the sections were then ground thinner and stained with picro-carmine. The central part then showed more or less *yellow* staining, microscopically visible (cf. p. 56).

For purposes of control a tooth with deep incisions made by grinding was placed in calf blood, renewed every week and mixed with Ringer's solution, in which it was kept for two months. After being removed and washed in alcohol the tooth showed no colour change at all.

B. Intra Vitam.

The difficulties to overcome in this branch of the subject were the uncertainty as to how long a time the experiment would require, the impossibility of maintaining efficient insulation, and the circumstance that in the case of exposed dentine, which is without protection, the invasion of diffusible salt (= diosmosis) is possible to so great an extent as to prevent hemal colouring matter diffusing out. A diffusion of plasm only would perhaps hardly be perceptible in this case.

First Experiment.

The subject was a lady of twenty-five years of age, and on the buccal side of one of her upper premolars a cervical cavity was ground square against the bottom (as

for inlay). Out of thin platinum plate a ring was made that exactly fitted the inner walls of the cavity, and, after grinding away the protruding parts of the top, a platinum cover was soldered on to the ring so as to stretch over the buccal side of the tooth. In the cavity thus enclosed, salt and sugar was placed, dissolved in a drop af water, and the platinum-roof was cemented over the cavity of the tooth. Of course, no cement was allowed to fall into the cavity.

Tests were made with sodium chloride, saltpetre and cane sugar.

Sugar — three, six and eighteen days respectively. No result.

Common salt — three, six and twenty-four days respectively. Result: After three and six days no change. After the twenty-four days' test the cover was very difficult to remove, and when the cavity was opened, a brownish-black material was to be seen on the bottom and walls. The author was unable to identify this material with blood, as, on account of a slight lack of caution in removing it, some blood unfortunately penetrated into the cavity from the papilla. In itself these tests probably had no value, and are mentioned only to show the methods. As the cover was hermetically sealed with cement and every trace of salt had disappeared on opening the cavity, the entrance of inorganic salts into exposed dentine, at any rate under favourable conditions, must be regarded as a fact. Compare the lipoid theory of OVERTON and its criticism in HÖBER [1]. According to this theory no organic salt can enter living tissue, *i. e.*, membranes of cells. During a conversation the author had with Prof. OVERTON; the latter expressed himself as being of the same opinion in so far as it applies to dental tissue.

---

[1] HÖBERT, 1911 p. 181. cf. with OVERTON, Pflügers Arch. 92. 1902. 115.

Second Experiment.

A girl, twelve years old, had at the beginning of the experiment her three upper premolars absolutely free from caries after washing with alcohol. The first left premolar had mesially in the fissure a pointlike cavity. Over the second premolars, which were standing perfectly free on account of the extraction of the first molars, small rubber sacks — s. c. »preservative seals» — were fixed, the right one being filled with about 1 cgm. of cane sugar and water, and the left one with about 0.1 cgm. of NaCl and water. Before the experiment a little grinding was done disto-buccally with carborundum and a small needlelike drill-hole was made mesially in both fissures. The sacks were attached to the teeth on the 12th August 1913 and the result, which was recorded in diary form, was as follows: —

1913	*NaCl.*	*Sugar.*
17/8	The sack torn. No change.	Extremely weak dark colouring of fissure.
18/8	»	»
21/8	The fissure slightly darker.	As before.
23/8	The fissure coloured weak brown. Small black spots here and there in depressions in the enamel.	»
27/8	The fissure distinctly, but weakly, »carious» in its whole length. The buccal spots appear darker.	The fissure clearly but weakly brown.
29/8	No change.	No change.
4/9	The sack was fastened with gold wire round the neck.	The sack was fastened with gold wire.
6/9	Distinctly appearing fissure caries.	Faintly appearing fissure caries.
10/9	The same condition.	The same condition.

14/9	The sack was filled with 2 cgm. lithium-chloride + aqua [1].	The sack was filled with NaCl.
16/9	*Distinct black fissure caries. Numerous dark spots lying in pointlike histological defects on the enamel.*	The fissure slightly darker than before.

The sack containing NaCl often broke and the other one frequently fell off, which rendered the tests difficult. Neither the incisions nor the drill-holes showed caries, excepting, as said, the fissure itself, *which confirms the fact that the crown-caries in the first place is dependent upon the presence of congenital histological entrance and not of acquired traumatic defects.* The sack containing salt sat much the tighter, *for the salt etched (resorbed) the gum round the neck 1—2 mm. upwards* and thus made it possible for the sack to press higher up against the root of the tooth. This destroying of the gum cannot be ascribed to the effect of the rubber, as the rubber sack containing cane sugar had no such effect. The adjacent teeth suffered no change at all during the experiment. That the tooth tested with sugar was not so strongly attacked is to be ascribed to the unequal osmotic strengths of sugar and NaCl in the same concentration but more to the fact that, during several experiments, the sack containing sugar did not remain in position. The black points slowly disappeared afterwards, but the fissure-caries remained, although weaker. The gingiva also grew back again. Such a test can, of course, only be successful in the case of very young teeth.

---

[1] The maximum dose of lithium-chloride is 25 centigrammes.

Artificial Diffusion Through Mucous Membrane.

On account of the effect of NaCl upon the gingiva, as related in the previous experiment, the following tests were made: —

In the mouth of a lady of twenty years, the crown of the first upper premolar being destroyed, the root being, however, still there, the author inserted an appliance which consisted of a block of vulcanite. This was fastened to the canine tooth and the second premolar by means of clasps. The appliance covered a surface of about 4 cm.^{2} of the outside of the gingiva. On the wall of the appliance turned towards the gingiva a cavity was bored of a definite geometric form and of about 3 mm. depth. In this cavity 1 cgm. of different salts and cane sugar was laid and the appliance was put in its place. The effect can be seen from the table given upon next page.

It is to be remarked that three days were allowed to pass between each test, but it is nevertheless possible that the gingiva in the end became affected.

As can be seen, NaCl is an extremely powerful caustic for the gingiva, when remaining for some time in the mouth. The diosmosis (?) was too strong for the mucous membrane. The rest of the table speaks for itself. It may also be added that the experiment was quite painless, and that no taste of salt was even perceived, except immediately after the insertion, when a weak taste was discernible, which is very remarkable, and is probably to be ascribed to an immediate dissociation. (Cf. KAHLENBERG [1]). It can also be accounted for by the saliva in this mouth containing a high percentage of salt (chlorine). By these tests the author obtained proof confirming the fact that NaCl resorbs the gingiva, which is of importance as regards the arising of root caries, and, further, that a mixing in a colloid substance increases the effect, when otherwise neutral salts

[1] ref. HAMBURGER, III 1904 p. 169.

Table

showing results of experiments to ascertain effect of certain salts upon the mucous membrane of the mouth: —

I. Nature of the Insertion.	II. Duration of Experiment.	III. Swelling of the Gingiva.	IV. Appearance of the Gingiva.
1. No insertion (the appliance only).	3 days.	Slight.	Normal.
2. Sugar and cocoa-nut oil.	3 days.	A little greater than in 1.	Normal.
3. Saltpetre + cocoa-nut oil.	3 days.	The same as in 2.	Normal.
4. Ferrum pulv. + one drop of water.	3 days.	Slight.	Reddish colouring.
5. NaCl + cocoa-nut oil.	1 day.	Pronounced.	Reddish colouring. Bleeding.
6. NaCl + cocoa-nut oil.	3 days.	Pronounced.	Much bleeding on exposure. The membrane open here and there.
7. NaCl + cocoa-nut oil.	1 week.	Pronounced.	Appearance of ulceration.
8. NaCl without oil.	3 days.	Slight.	Reddish colouring.

effect a sinking of the osmotic pressure of hydrophile colloidal suspensions [1].

Summary.

1. The author succeeded in getting defibrinated blood to diffuse through thin sections of teeth.

2. The author succeeded in producing *in vivo* the small black-brown points in the enamel characteristic of very early caries. The typical feature of these tests were:

a) That minimum black spots appeared here and there over the whole crown.

b) That all of them were standing in connection with histological defects and pointlike depressions of the enamel.

c) That NaCl used in the test strongly resorbed the gingiva.

Criticism.

1. The result of the first tests *(in vitro)* belongs rather to the tests showing the osmotic properties of the dentine.

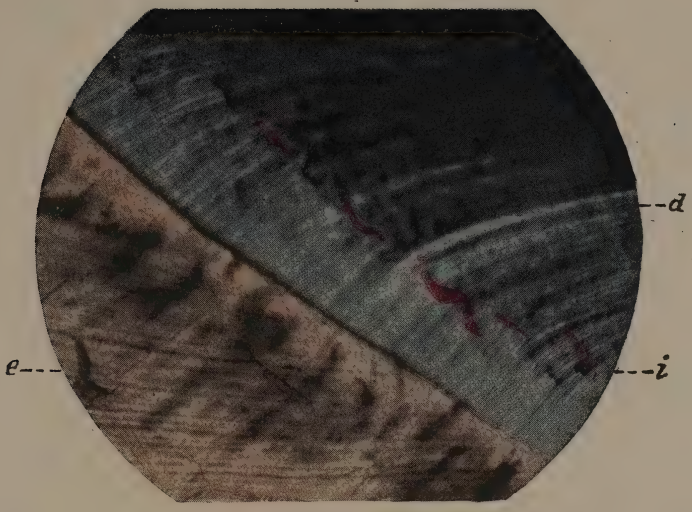
2. The specific way of trying to generate caries certainly does not result in producing *open* caverns, but is in so far remarkable that it gave — in spite of being a test *in vivo* and belonging to a disease which generally takes a long time for its development — a positive result.

3. The impossibility of producing artificially open caverns upon dentine or enamel is an indirect proof that this process is closely associated with life.

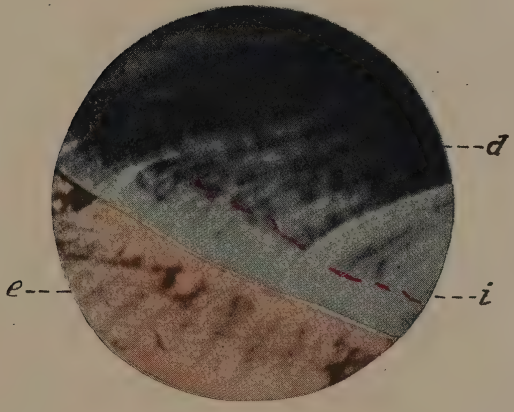
---

[1] cf. HÖBER, 1911 chapt. IX.

Plate VIII.



175 × enlargement.



79 × enlargement.
e = enamel; *d* = dentine; *i* = interglobular spaces.

Micrograph

of a section of Canin. sup. dextr. injected *intra vitam* with fuchsine (1 + 100). The section is embedded in Canada balsam. Traces of methastatic fuchsine in the interglobular spaces.

CHAPTER IV.

Special Tests Intra Vitram.

Tests showing the Relationship between the Vascular System and the Liquid streaming in the Tubuli.

If a solution of 1 % methylene-blue or 0.1 % fuchsine, to which a drop of ethyl alcohol has been added, is injected with pressure through the apex of a tooth (v. BEUST [1]) having small initial carious points (primary caries), it will be seen microscopically immediately after splitting the tooth, that the pulp has taken an intense blue or red colour and that in the case of young teeth the stain has entered the dentine up to the dento-enamel junction and into the enamel. These stains do not seem to be able to penetrate young caries-canals and new dentine, but other dyes, *e. g.*, gentian violet, stain the carious dentine. These tests, which do not concern the problem as to whether or not a conversion of matter takes place in the enamel, seem to confirm to a certain degree the experiments and claims of MORGENSTERN [2], CAUSH [3], BOEDECKER [4], v. BEUST [1], a. o., namely that the lymph or serum-system of the body terminates in the enamel.

In order, however, to obtain full evidence of any circulation of fluid that may take place from the pulp to the

---

[1] v. BEUST, Arch. f. Zahnheilkunde 2. 1912.
[2] MORGENSTERN, Schw. Vierteljahrschr. 1901. 185.
[3] CAUSH, Dental Cosmos 1905.
[4] BOEDECKER, Dental Review 5. 1915.

enamel in the dentinal tubes and the hard parts of a tooth, experiments of quite another type must be undertaken.

For the experiments in question intact teeth of young individuals were selected, but such teeth as were destined for extraction on account of irregular position. Local anaesthesia having been produced[1], a canal was drilled with a fine bur from the facial side up to the living pulp, great care being observed on approaching the pulp cavity. The hole thus formed was cleaned, and through it various, previously sterilized, stained fluids of one per cent. strength, such as methylene-blue, methylene-green, gentian violet, and fuchsine, were pumped into the pulp cavity by means of a syringe having a needle that tightly fitted the canal. Prof. OVERTON recommended the use of methylene-blue for this purpose, but for technical reasons this stain did not prove suitable. A few drops of the stain having been injected, the needle of the syringe was cut off tight against the tooth, and the bore-hole with the part of the needle left in it was sealed with glass-wax. Periods varying from 5, 10, 15 to 20 minutes were allowed to pass[2], after which the tooth was extracted and carefully cleaned of all external trace of colour. The apex was sealed with glass-wax and the tooth immersed for twenty-four hours in Ringer's solution. It was then taken out, cut into very thin sections and embedded in Canada balsam. Several such experiments were made. It was ascertained that under special conditions the stain had been conveyed to the dento-enamel junction, that is to say, the younger the tooth the denser and richer was the colouring, which, of course, was also influenced by the time allowed to elapse before extraction. New-dentine and carious dentine were, according to my observations, impermeable (or only slightly permeable) to the stain. It should be noted that the colouration did not take place through the medium of the constituents of the dentinal tubes so that all the organic tissue there was stained, but

[1] *Albromin* preferable for this purpose.

[2] There is no risk of poisoning.

scattered patches of stain appeared here and there in the tubules, the organic tissue between these remaining uncoloured. A sure proof that a transport, and not a diffusion of colour had taken place.

A photo-micrograph of a section from a tooth treated in such a manner is reproduced here (Plate VIII) in two enlargements 79/1 and 135/1 respectively. (The original is in the author's possession and may be inspected at any time.) The section is taken from the canine of an individual seventeen years old, and the tooth had been erupted about two years. The photograph shows in its right upper field the layer of dentine and in its left lower one the enamel portion. In the interglobular spaces traces of fuchsine are observed, which stain was used in this case, giving in general the best result. Both methylene-blue and methylene-green proved useless on account of the dentine in the section, embedded in Canada balsam, taking on a blue-green tint. The photo-micrograph shows that there is no trace of colour in the dentinal tubules between the pulp and the dento-enamel boundary, whereas there are, as will be seen, small patches in the interglobular spaces.

By what paths has the colour reached the interglobular spaces? Two paths are conceivable: 1) from the pulp through the tubules, 2) from the bore-hole laterally. The latter assumption is purely theoretical. It would presuppose a mutual peripheral communication between these spaces, of the existence of which we lack knowledge. There is no evidence whatever in favour of this supposition. The patches of colour in the interglobular spaces are not connected with one another, but detached. We must, therefore, accept the first alternative, *i. e.*, that the colour has reached the interglobular spaces from the pulp through the tubules, a claim, *which is, as already shown, also confirmed by other similar experiments,* where it was shown that solitary patches of colour are always to be found in the

tubules. From these experiments and from the photomicrograph we are justified in making the following

Resumé.

1. There is a circulation of fluid from the pulp-cavity to the dento-enamel boundary — the interglobular layer — and in certain cases, through offshoots, into the enamel, for it follows *eo ipso* that a fluid must be the agent by which the transport of colour is effected.

2. This circulation is more active the younger the tooth.

3. The fluid does not seem to pass through secondary dentine and carious dentine, these kinds lying outside the circulation.

4. Inasmuch as the stained fluid stopped in this case in the interglobular spaces, no trace of colour being seen in the dentinal tubes, we are entitled to claim that the interglobular spaces have a physiological function of another nature.

5. The interglobular spaces have commonly been regarded as »Uncalcified dentine» (s. *inter alia* HAUS [1]).

It would be strange, however, if the fuchsine, in this case transported by what we may designate the dentine-fluid, should just come to a stop in the *uncalcified* dentine. Moreover, what do these authors mean by »uncalcified» dentine? Partly calcified or not at all calcified? It must be assumed that *organic* constituents are present in either case. But if the organic matter in the interglobular spaces is stained *in vivo*, why is not all the organic tissue passed by the staining fluids stained, at the same time *e. g.*, the adjacent organic constituents of the dentinal tubes, which otherwise readily permit of staining by fuchsine? It is, therefore, evident that the fuchsine appears in these dentine

---

[1] HAUS, 1917 p. 135.

formations not as a true staining-material but as a product of sedimentation or as a cast-off product (metastasis).

The supposition, on the other hand, that the interglobular spaces are identical with »uncalcified» dentine is based upon the observation that during its embryological (»ontogenetical») formation the dentine exhibits similar formations at the border of the dentine-forming layer of odontoblasts. We are justified in asserting that this pretention is loose and unproven, for these interglobular spaces are also to be found in teeth where the pulp has contracted itself into the root and all the crown-dentine has been transformed into secondary dentine, in which complete calcification(?) has taken place. Do these spaces permanently remain uncalcified? Or is the whole conception wrong? Everything speaks in favour of the latter alternative, and we are probably justified, as already mentioned, in asserting that the interglobular spaces in the dentine represent a physiological organ for the tooth, the real function of which is unknown as yet. This assumption does not stand in opposition to the opinion that the interglobular spaces perform a function in the new-formation of dentine. MORGENSTERN[1] regards the spaces as »paths for the lymph». So much should, however, be said — that the intruding stain must be regarded by the tooth as a hostile alien that is to be rapidly removed, isolated and carried away. It is then transported to this place.

The expression »Interglobular spaces» seems also unsuitable. It may be questioned, whether these »spaces» do not form peripheral *spiral-like lacunae, filled with liquid.* The spiral-form is shown by the even staining, and the even staining speaks for the presence of a *liquid.*

6. *The presence of a fluid system from the pulp to the dento-enamel boundary, thus established, constitutes a*

---

[1] MORGENSTERN, D. M. f. Z. 10. 1909. 717.

fact that confirms one of the fundamental principles in the osmotic theory.

Critique.

It is to be noted that the fuchsine readily colours the organic tissue of the tooth, and, hypothetically, it should more easily penetrate moist, living organic tissue than dry, dead tissue. If the stain had pressed its way up, it must have coloured the path it traversed. Consequently all attempts to regard this test as a pure staining experiment or diffusion phenomenon, collapse upon the fact that the colour isolated itself in this case in the interglobular spaces. If all the contents of the dentinal tubes had been coloured, the matter would have been debatable, and only the short exposure time during which the colour was spreading would have spoken in favour of a metastatic process by fluidal transport. The possibility that on injection the colour immediately made its way to the sides of the drill-hole and thence direct into the interglobular spaces, and not *via* the dentinal tubes, on the supposition that these spaces are in mutual communication, is improbable for the reason that a pure diffusion of colour must then have taken place, which would in at least some places have extended from the interglobular spaces into the tubes. In such a case the colouring would also have had quite another character, namely a thick and deep stratum of colour nearest the drill-hole thinning outwards. This is not the case. Indeed, as repeatedly emphasized, small spaces of colour have been found here and there in the dentinal tubules in the case of other fully analogous experiments, clearly showing that the colour had passed that way before it was deposited peripherally.

Table 1

showing the relationship between Sugar and Common Salt and the Oral Acidity.

SUGAR. (Piece of Sugar chewed)			
Person	Reaction before the test	Time	Reaction after the test
40 years	weakly positive	5 min.	sharply positive
»	—	10 »	positive
»	—	30 »	neutral
»	—	the follow. day (in vitro).	positive
22 years	strongly negative [1]	5 min.	weakly negative
»	—	10 »	negative
»	—	30 »	strongly negative
»	—	the follow. day (in vitro).	negative
25 years	neutral	5 min.	positive
»	—	10 »	neutral
»	—	30 »	weakly negative
»	—	the follow. day (in vitro).	positive
25 years	positive	5 min.	positive
»	—	10 »	positive
»	—	30 »	weakly negative
»	—	the follow. day (in vitro).	strongly positive

[1] HCl assumably present.

Resumé of Table I.

Sugar in vitro is a very weak acid-former. It is, consequently, still weaker in the mouth [1]. That the reaction of the saliva *in vitro* is always positive the following day, means assumably that the alkaline fermentation of the saliva has neutralized the weak acid possibily formed. This fermentation is a proof that Black's gelatinous membrane, under the protection of which the fermentation of acid would hypothetically proceed and which is to be regarded as a sedimentation-product from the saliva, also will be fermented and destroyed by microorganisms. Thus from the very animal »protecting plaque» alkalines are formed, which probably quite neutralize or in any case counteract possibly formed acids of microorganic origin, produced upon the teeth.

Resumé of Table II.

1. For common salt to be able to form acid the presence of free H-atoms is necessary.

2. In positive or neutral saliva free H-atoms are sometimes present in sufficient quantities, sometimes not. In negative saliva free H-atoms are always present, because organic acids give off their H-atoms by dissociation. Consequently *weakly acid saliva will by addition of NaCl successively terminate in strongly negative.* Positive or neutral saliva, on the contrary, will sometimes become negative, sometimes not. The tests with acidulated saliva confirm this conclusion.

3. Sugar together with NaCl seems to give a *positive* reaction rather than a negative one.

4. The microorganisms in the mouth are able to neutralize all organic *acids,* with the exception of the strong »mineral» acids, by causing fermentation of the

[1] cf. Hopwell-Smith, Dental Cosmos 9. 1915.

Table II.

Common Salt.

Person	Reaction before the test	Time	Reaction after the test.
40 years	weakly positive	5 min. (in mouth)	positive
»	—	5 » (in vitro)	weakly negative
»	—	10 » (»)	negative
»	—	30 » (»)	strongly negative
»	—	the following day	positive
22 years	positive	5 min. (in mouth)	neutral
»	—	5 » (in vitro)	weakly negative
»	—	10 » (»)	negative
»	—	30 » (»)	negative
»	—	the following day	positive
25 years	positive	5 min. (after adding salt)	strongly negative
»	—	10 » (»)	» »
»	—	30 » (»)	» »
»	—	the following day	» »
22 years	strongly positive	5 min. (after adding salt)	positive
»	—	30 » (»)	»
»	—	the following day	»
39 years	weakly negative	5 min. (after adding salt)	strongly negative
»	—	the following day	» »
21 years	positive	30 min. (after adding salt)	positive
»	—	the following day	»
40 years	neutral	30 min.	neutral
»	—	the following day	positive

saliva, in that way producing alkalines — a wise arrangement of »the system of nature».

Criticism.

Sugar has since ancient times been regarded as an incontrovertible cause of caries. It is now proven that sugar is a very weak acid-former. Consequently it must be on account of another property that sugar may produce caries.

The litmus paper gives no reliable clue as regard finer differences in the reaction, but only as regards the more rough ones. Positive = alkaline; negative = acid saliva.

CHAPTER V.

The Saliva.

The general properties and function of the saliva are rather well treated in our text-books [1]. Here the saliva will be regarded from some points of view that are not so well known.

Cryoscopy of the Saliva.

By cryoscopical researches we mean the common way of determining the osmotic pressure of a liquid by calculating the freezing-point.

From an osmotic point of view we may divide the liquids of the body into constant and inconstant ones. The blood and the lymph are constant, the blood having practically a constant pressure of 6.7 atmospheres (VIOLA, HAMBURGER, ARRHENIUS), over 8 atm. according to HÖBER [2]. Only in certain maladies and in cases of great disturbances of the normal state, *viz.*, certain forms of nephritis and pregnancy, etc., does the pressure substantially diminish or increase. Very small deviations during a non-pathological state also: rising after eating, sinking after drinking large quantities of common water.

The inconstant liquids are the urine, the saliva, etc.,

---

[1] s. MICHEL, Deutsche Zahnheilkunde 10. 1909,
BRUGNATELLI, 1913 p 92. and
MARSHALL: The American Journal of Physiology 1915, 1916, 1917 the latter cf. with
SHEPHARD GIES: Journal Allied Dent. Soc. 1916.

[2] Cf. HAMBURGER I 1902 p. 503, with HÖBER: 1911 p. 74.

i. e., liquids from the secretory glands. Their osmotic pressure changes from hour to hour. To show the difference between constant and inconstant liquids the following is a good test:

Into a horse several pints of a diluted green ferro-sulphate solution was injected. After some minutes all the glands standing in the service of external secretion were at work, and the urine, fæces, saliva were all coloured green. What is the explanation of this? The blood will keep its osmotic equilibrium at any price unchanged, and, therefore, immediately eliminates the injected salt by the external glands (HAMBURGER I 1902).

Salt (NaCl-solution) has been injected in the jugular vein of a dog and it was shortly afterwards found that the percentage of chlorine in the saliva had very much increased (NOVI) [1].

The cryoscopical investigations of the saliva found in the literature are very few [2]. According to the figures published, saliva has an osmotic pressure varying between freezing-points 0.11° and 0.27° C. (that of blood is 0.56° about). These figures may be criticized as follows: —

1. The authors do not state the temperature and therefore we can assume that the figures are the result of pure cryoscopical calculations, *i. e.*, the osmotic pressure at 0° C., but a figure so obtained is much too low, as the osmotic strength increases rapidly with rising temperature. The true figure is obtained first after fixing the »dissociation-constant» *which certainly varies in different salivas.*

2. For common cryoscopical tests in general it is necessary to have a quantity of 10 to 15 grammes. To get this quantity of saliva one must »collect» at different

---

[1] Cf. HAMBURGER, II 1904 p. 425; cf., also, BUNTING and WIXON, The Journal of Nat. Dent. Ass. 1. 1917. 82.

[2] Cf. FANO and BOTAZZIO, Archiv Ital. de Biol. 26. 1896. 45.
BÖNNINGER, Arch. für ex. Pathologie 1903 L. 76.
WOLF, Arch. de. Biol., XVIII. 2 p. 241.

times, which will give an incorrect figure. Furthermore, saliva gathered in that way, often from different persons, loses during the time many of its qualities. »It was subsequently stated by the writer that comparisions of saliva, the samples of which have been collected under different conditions, are inadmissible since such procedure ignores entirely physical influence.» MARSHALL [1].

3. The two ways (that the author knows) of exactly measuring the freezing-point of very small quantities are based upon a special principle, the s. c. »Hematocrite-method» [2].

Practical application: A fixed quantity of defibrinated blood is centrifuged in a graduated thermometer-tube, and the height of the pillar being found is measured. Exactly the same quantity of blood is then centrifuged with NaCl-solutions of different concentrations. That solution which gives a pillar of corpuscular sediment of quite the same length as the undiluted blood is isotonic to the blood in the test.

The other method is the »Micro-cryoscopical method» [3].

Practical application: A minimal-drop of the saliva in question is shut up in a fine capillary-tube. The temperature at which the drop is quite clear, *i. e.*, in the moment the last split of ice is melting, is the freezing-point of the test. —

4. It may further be emphasized that in order to get the freezing-point mentioned above, the samples of saliva are assumably taken from saliva during *normal states.* Of course, what we want are samples of saliva from people with devastated mouths, and from people who daily consume large quantities of salt, sugar or medicine, the investigation being made immediately after the consumption.

5. There is further a great difference in the concen-

---

[1] MARSHALL, 1917 p. 212.

[2] HEDIN, Pflügers Arch. 60. 1895. 360; cf. KOEPPE: Du Bois-Reymonds Arch. 1895. 154.

[3] DRUCKER & SCHREINER, Biol. Centralblatt 33. 1913.

tration of saliva at different times of the day, before or after meals, in the day or night. In the case of people who drink very little water the morning saliva is very concentrated.

6. The osmotic strength of a liquid does not only depend upon dissolved salts and chemical materials. Upon cryoscopical test the gases will volatilize on freezing, and in that way deprive the liquid of an essential osmotic factor. (The gases are certainly undervalued as to their power of increasing the osmotic pressure of a liquid. »General» opinions are not always axiomatic truths) [1].

7. Finally we can speak of a total osmotic pressure of the saliva and a local one, the latter being active under the influence of such colloidal substances as albumen, fat, mucin, etc., which are mixed with salts from the food, and act on places where the process can continue undisturbed. For, like all chemical and physical processes, osmosis ceases or proceeds with difficulty when subject to disturbing influences.

The Scientific Proofs for the Assumption that on Certain Occasions the Saliva Can Obtain Higher Osmotic Pressure.

1. The law of HEIDENHAIN:

»With the increase of the velocity of the secretion the percentage of inorganic salts in the saliva also grows up to 0.6 % (according to WERTHER, up to 0,77 %)». This law is also confirmed by other investigators (s. HAMBURGER [2]).

---

[1] Cf. later on in this chapter the difference between experimentally found figures of the freezing-point depression of saliva and the »official» figures and further as regards the influence of carbonic acid upon blood s. HÖBER, 1911 p. 29.

[2] HAMBURGER, II 1902 p. 423.

2. From the tests of NOLF [1] we find that the sinking of the freezing-point of the saliva depends almost entirely upon the *salts* of the saliva.

3. By the injection of a strong NaCl solution in the jugular vein of a dog, it was shown that the percentage of chlorine in saliva grew considerably, and more swiftly than in the plasm. (Tests by NOVI [2]).

4. After injection of strong salt-solutions (2%), it was shown that the salt-percentage of the saliva had increased. By injection of diluted solutions the total quantity of saliva was increased, the percentage of salts diminished. LANGLEY and FLETCHER [3]). The same investigators have shown that after injection of lithium-citrate in the blood even the first secreted drop of saliva contained trace of lithium-citrate.

5. The gastric juice, which enters the mouth in greater or smaller quantities, has commonly a greater osmotic pressure than the saliva, as varying between 0.16—0.72, and assumably it is that, with a high percentage, which rises to the mouth.

6. The normal freezing-point of the saliva (freezing-depression (Δ) under 0° C.) varies between 0.09°—0.24° C. (BÖNNIGER l. c.), between 0.11°—0.27° C. (WOLF l. c.) etc. cf. HÖBER [4].

Conclusion.

From these laws and experimental facts it can be *theoretically* concluded that as regards osmotic pressure the saliva differs from time to time. We know, moreover, that civilized people consume enormous quantities of such

---

[1] NOLF, Travaux du Labor de Physiol. Liège 6. 1896—1901. 225.

[2] ref. HAMBURGER, II 1904 p. 425.

[3] LANGLEY & FLETCHER, Phil. Transactions Royal. Soc. 1889. 109.

[4] HÖBER, 1911 p. 533.

chemical matters — crystals in solution — as call forth an increased osmotic pressure of a liquid, *i. e.*, crystalline sugar, salt, saltpetre, salts of mineral-water, medical preparations, etc. The averment that at certain intervals saliva can obtain an osmotic pressure greater than that of the blood is thus on account of theoretical reasons free from objections.

Proof that the Saliva can acquire a Pressure nearly equal to that of Blood.

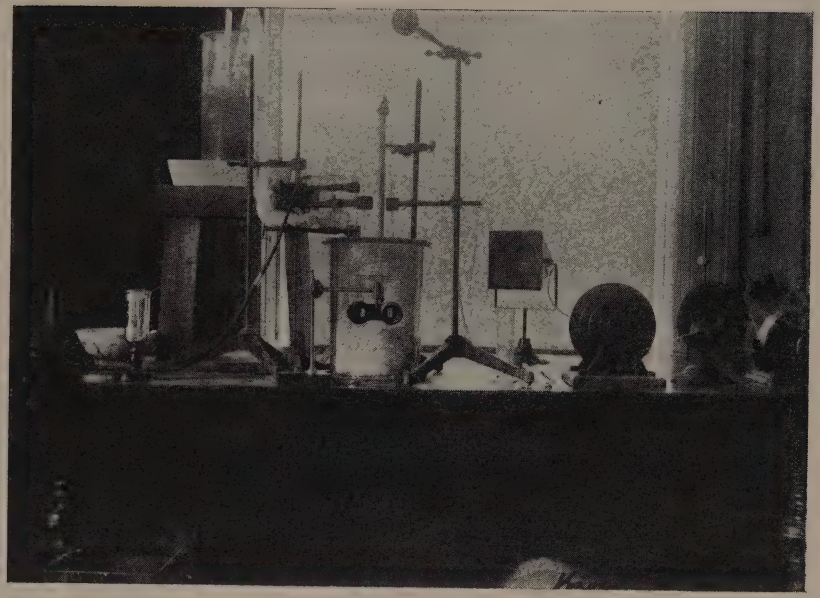
A simple practical method of determining whether the saliva at certain intervals may be osmotically hypertonic to blood (plasm, serum) is based upon the fact that a hypertonic solution causes a shrinkage of the red corpuscles, while a hypotonic one causes them to swell, *i. e.*, produces hemolysis [1]. If thus blood is thrown into a certain liquid, we either get hemolysis or we do not. If in a state of isotony, *i. e.*, an equilibrium of pressure, no colouring appears either.

The test is carried out in the following way. Take defibrinated horse blood, which has the same osmotic pressure as human blood, and let a sediment be formed. The plasm is drawn up by means of a pipette. It is of advantage to have the blood in a rather fine test-tube. Fresh saliva (1—3 cm.^{3}) is also placed in another test-tube and in this we allow one drop of the *blood-sediment* to fall, but in such a way that the blood comes under the froth, which is always formed on the surface. After careful shaking, the test-tube is allowed to stand until the following day. If the liquid over the sediment is a clear red, hemolysis has set in and the liquid is hypotonic, but if it is yellow or not red the liquid is hypertonic *i. e.*, a shrinkage of the erythrocytes has taken place.

The author's own results show so far that *normal*

[1] S. Hamburger, I 1902 p. 185.

Plate IX.



Micro-cryoscopical Apparatus.
(After DRUCKER and SCHREINER).

saliva is nearly always hypotonic to blood. But while eating sweets or salted food, or after having taken medical powders or crystals, and sometimes also in some other cases, *the saliva exhibits hypertony or possibly full or nearly full isotony to the blood.*

Critique.

Hemolysis, or the expulsion of the hemoglobin, does not occur exactly at the limit of isotony, but *under the limit*, as the red corpuscles of the horse offer less resistance than those of the human blood. If thus the human hemoglobin is normally expelled first in 0.45 % NaCl solution, the hemoglobin of the horse is expelled already in 0.68 %. (According to HAMBURGER and DE VRIES human hemoglobulin hemolyses in 0.58 % NaCl-solution) [1]. If in the experiments mentioned above no hemolysis is seen, *we really only know that the saliva in the test has higher osmotic pressure than* 0.68 % *NaCl* [2]. But this naked fact is itself an exellent indication, when we remember that the common cryoscopical tests have given the saliva a freezing-point depression of about 0.1°—0.2° C. only, corresponding to one-third to one-fifth of the osmotic pressure of the blood. If the saliva can augment its osmotic pressure three-four times, it is obvious that it is able to increase its pressure four-five times its original pressure (= 0.4°—0.9° C.), and then it is already greater than that of the blood (= 0.56°). We can also observe in the test under discussion that sometimes the liquid (saliva) standing over the sediment is very clear, sometimes not so clear, which shows that we sometimes have more or less hypertonic saliva. On the other hand, in repeated consideration of the known fact that large quantities of

[1] HÖBER, 1911 p. 72, cf. with HAMBURGER, I 1902 p. 165.

[2] The osmotic pressure of blood corresponds to about 0.90 % NaCl. The resistance of the red corpuscles varies during pathological states.

salts and sugar pass through the mouth, it is quite evident that a concentration of the saliva by 1 % NaCl or 10 % sugar is, for a longer or shorter time in the case of »civilized» people, a daily recurring fact.

For the purpose of obtaining conclusive experimental proof that the mastication of sugar or salts imparts to the saliva an osmotic pressure higher than that of blood, the following experiments were undertaken for my account at the University Institute of Physiology in Lund.

After the mastication of the different substances (Experiment I — Cane Sugar; Experiment II — Common Salt; Experiment III — Acetyl-Salicylic Acid) samples of saliva were taken from time to time, only a minimum quantity being selected each time. The freezing-point depression was determined by the micro-cryoscopical method given by DRUCKER and SCHREINER[1], after which the osmotic pressure was calculated by formula $P_{osm} = \Delta \times \frac{22.35}{1.85}$. This method aims at fixing the temperature at which a minimum drop of frozen saliva enclosed in a thin-walled capillary-tube becomes fully clear, that is to say, at which the last ice-particle melts (s. Plates X—XII and XIII—XIV).

Resumé.

1. These experiments supply an infallible proof of the truth of the hypothesis which constitutes one of the corner stones of the osmotic theory, viz. that consumption of sugar and salts (sodium chloride) imparts to the saliva an osmotic pressure much higher than that of blood.

2. During the experiments this pressure rapidly declined, the saliva exhibiting a remarkable tendency to come quickly pouring into the mouth and hence reducing the concentration — in the case of cane sugar already

---

[1] DRUCKER and SCHREINER, Biol. Centralblatt 33. 1913. S. also HANS MEYER, 1917.

Plate X.

Test I. **Cane Sugar.**

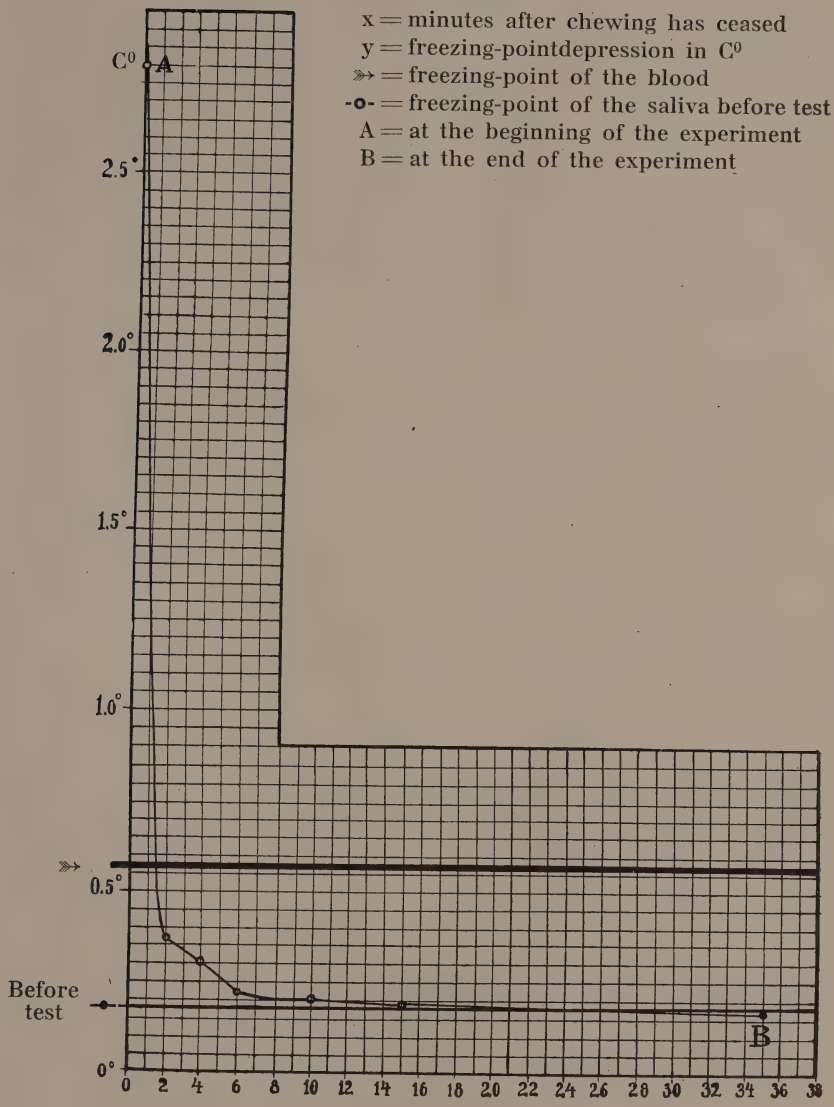


Plate XI.

Test II. **Sodium Chloride.**

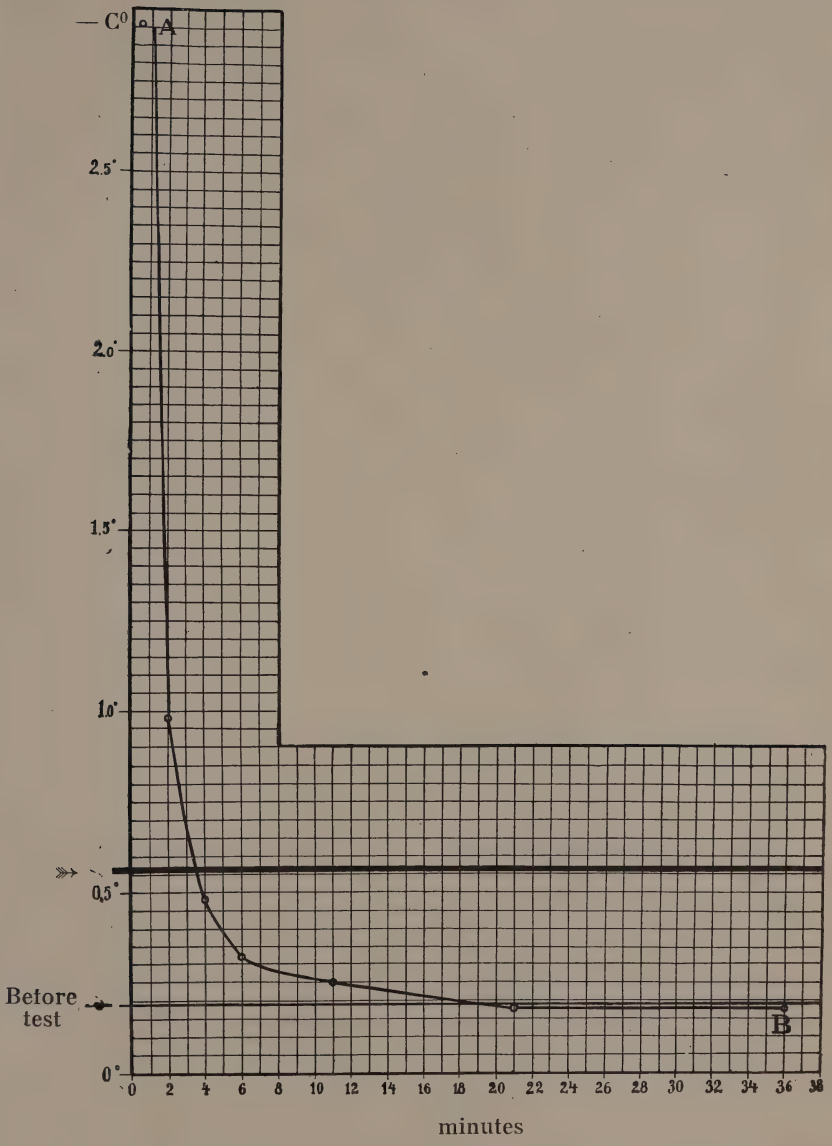
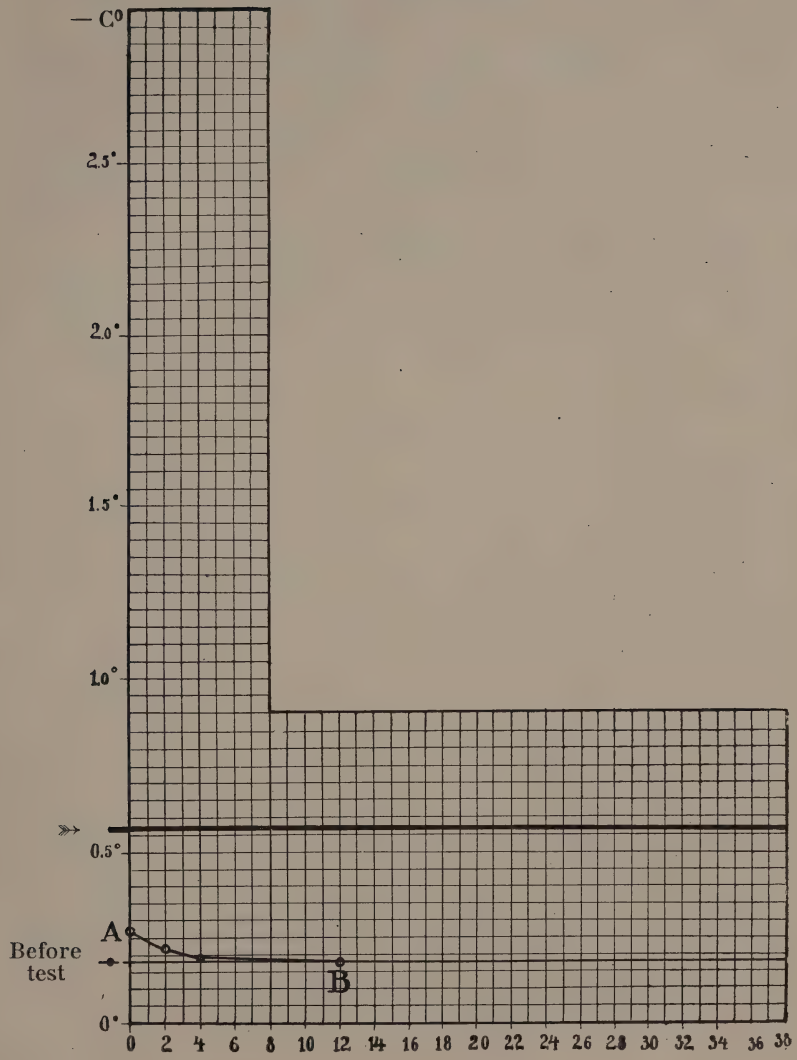


Plate XII.

Test III. Acetyle Salicyle.



after 2 minutes, common salt after 4 minutes. This circumstance explains why all the freezing-point calculations hitherto made of the saliva have given such low figures.

3. One gr. of table salt was found to give the saliva an osmotic pressure higher than 50 atmospheres — more than seven times the osmotic pressure of blood.

4. About 6 gr. of sugar, 1 cube, gave an osmotic pressure of 33.82 atmospheres — five times the osmotic pressure of blood — immediately after the sugar had dissolved.

5. As regards acetyl-salicylic acid this is only slightly soluble in water (saliva). Other readily soluble medical preparations, bromic salts, etc., had given a pressure analogous to that of common salt.

Critique.

1. Compared with the so-called Hematocrite method the Microcryoscopical method possesses the advantage that exceedingly small quantities of saliva may be used. Furthermore, it requires considerably less time, and, being a purely physical and chemical method, does not give rise to those inaccuracies always associated with physiological methods. On the other hand the hematocrite method is superior, giving the *total* osmotic pressure. For the present case, this is of no importance, as the inactive pressure of the sample of saliva, which would then be due to the presence of some substances possibly able to dissolve lipoid, is infinitesimal in relationship to the total osmotic pressure. A series of control tests showing the accuracy of the method was made with various solutions of glucose, from which (Table X) it can be seen that the deviations between the computed freezing-point and the freezing-points found by the micro-cryoscopical method are of an unessential nature.

2. A comparison between the result found by experiment and the conditions in practice or in life shows

Plate XIII.

Table X.

The accuracy of the micro-cryoscopical method is given by the following check-tests, fixing the Δ of solutions of glucose in different concentrations.

Solution of glucose 1	calculated $\Delta = 0.80°$	Received $\Delta = 0.79°$
» 2	$\Delta - 0.60°$	$\Delta - 0.62°$
» 3	$\Delta - 0.53°$	$\Delta - 0.53°$
» 4	$\Delta - 0.40°$	$\Delta - 0.40°$
» 5	$\Delta - 0.27°$	$\Delta - 0.27°$
» 6	$\Delta - 0.27°$	$\Delta - 0.26°$
» 7	$\Delta - 0.00°$	$\Delta - 0.01°$

Test I. Cane Sugar.

The 20th March 1918.

Object: 20-year's student; teeth without caries; previous meal at 10 p. m. Blood = 6.7 atm. or 0.56° Δ.

Time	Saliva-test	Δ = — C°	Osm. Pressure
12,30 p. m.	1	0.18°	2.17 atm.
12,43-45 p. m. 2 pieces of sugar chewed; lively salivation			
12,45 p. m.	2	2.80°	33.82 atm.
12,47 »	3	0.37°	4.47 »
12,49 »	4	0.30°	3.62 »
12,51 »	5	0.22°	2.65 »
12,55 »	6	0.20°	2.42 »
1,00 »	7	0.19°	2.30 »
1,10 »	8	—	—
1,20 »	9	0.17°	2,05 »
1,45 »	10	—	—

Plate XIV.

Test II. Sodium Chloride.

The 22nd March 1918.

Person: 20-year's student; teeth without caries; previous meal at 4 p. m.

Time	Saliva-test	$\Delta = -$ C°	Osm. Pressure
6,38 p. m.	1	0.19°	2.30 atm.
6,53-54 p. m. 1 grm salt chewed; lively salivation			
6,54 p. m.	2	> 4°	> 50 atm.
6,56 »	3	0.98°	11.84 »
6,58 »	4	0.48°	5.80 »
7,00 »	5	0.22°	3.87 »
7,05 »	6	0.25°	3.01 »
7,15 »	7	0.18°	2.17 »
7,30 »	8	0.18°	2.17 »

Test III. Acetyle Salicyle.

The 25th March 1918.

Object: 20-year's student; teeth without caries; previous meal at 4 p. m.

Time	Saliva-test	$\Delta = -$ C°	Osm. Pressure
6,50 p. m.	1	0.18°	2.17 atm.
6,52-53 p. m. 1 grm acetyle salicyle chewed; exceedingly lively salivation			
6,53 p. m.	2	0.27°	3.25 atm.
6,55 »	3	0.22°	2.65 »
6,57 »	4	0.18°	2.17 »
7,00 »	5	—	—
7,05 »	6	0.18°	2.17 »
7,15 »	7	—	—

that in the former case it is a question of a single experiment, e. g. a solitary lump of sugar, but in practice it is often a question of the successive consumption of a quantity of sugar, for instance, the contents of a bag of sweets: thus, a series of such experiments, where one quantity of sugar succeeds another. The same applies to salt: for the experiment a small quantity is used, but in reality albuminous food containing a high percentage of common salt is often consumed, this food giving off salt in the mouth for a relatively long period. The same remarks hold good in the case of the consumption of medical preparations.

3. Another aspect of the matter is that mouths containing carious caverns offer possibilities for the retention of food or other substances mixed with sugar, salts, or medical preparations, this implying that the saliva receives for a long period osmotic reinforcement. Hot drinks, of course, also increase the osmotic pressure.

4. The property of saliva with regard to viscosity *i. e.*, the character of being more or less a hydrophile-colloid solution must also influence the osmotic effect. That this is really the case the experimental figures show. If 6 grm cane sugar were taken in the mouth (maximum 1—2 cc saliva) the osmotic pressure would rise to a strength of more than 100 atm. even if the salivation should increase as much as four times the normal quantity. In a colloidal suspension or hydrophile-colloidal solution, however, the osmotic effect is in general reduced by the presence of sugars and electrolytes (s. HÖBER) [1].

Saliva and the Gases.

That saliva swiftly absorbs gases is a well-known fact. We have only to think of smoking, the pleasure and danger of which are based upon this fact. Those who have

[1] HÖBER, 1911 p. 363.

used gilding solution know how swiftly the acrid smell of potassium-cyanide is tasted in the mouth. Direct observations have shown, for instance, the increase of the rate of chlorine in the saliva by the injection of NaCl in the jugular vein.

That thus the saliva is a holder of gases there is no doubt (cf. MARSHALL [1]). That these gases must also increase the osmotic strength is obvious, as the osmotic laws are calculated and based just upon the properties of gases.

Strictly speaking all ions are to be regarded as gases.

»The osmotic pressure of a solution corresponds to the pressure which dissolved matter of the same molecular nature as gase or vapour in the same volume and at the same temperature develops» (VAN'T HOFF [2]).

What gases come into consideration?

The most important are carbon dioxide, chlorine, ammonia, hydrogen; further, metal and metaloid gases, *viz.*, lead, mercury, phosphorus etc.

Carbon dioxide.

The presence of carbon dioxide in the saliva can quantitatively be fixed by titration in the following way: —

If we have a mouth with high acidity (shown by litmus-paper), we let the person rinse the mouth carefully with *e. g.*, 50 gm. aqua dest. Divide the quantity obtained into two equal parts. Titrate the first with phenolphtalein; the second with methyl-orange. The difference (if there is any), multiplied by two, is thus the quantity of carbonic acid present. The explanation of this calculation is simple: methyl-orange does not give a decision in the case of carbonic acid, but phenolphtalein does, and consequently

[1] MARSHALL, 1917 p. 216.

[2] VAN'T HOFF, Zeitschrift f. Physik. Chemie I. 1887. 488.

the difference is just half the quantity of carbonic acid present.

Concerning the fact as to what degree of carbonic acid can be regarded as a harmful component, it should be remembered that this acid is a dissolver of salts of calcium, potassium, sodium, magnesium, which are otherwise indissolvable in water. A great surplus of carbonic acid will thus keep these salts in solution, which circumstance considerably increases the osmotic strength of saliva. Introduced in blood CO_2 very much raises the osmotic pressure (Experiments by KOVACS) [1]. The osmotic strength of the carbonic acid itself is great; greater than that of any other inorganic acid. The question is, however, if CO_2 can penetrate the carious matter or not? In the former case the effect of the »free» acid as an osmotic factor would be raised [2]. Only as a salt-builder could it then increase the osmotic pressure of the saliva. That many *organic* acids can enter is true. We know what a strong, swiftly passing feeling of pain the fruit-acids cause in decayed teeth. If carbonic acid is able to enter the roots of teeth after the outer membrane has been corroded, which is probable, this acid might be reckoned among the causes of the mysterious loosening of non-carious teeth.

Chlorine.

The presence of chlorine can be quantitatively detected by titration with *sol. nitr. arg.* Indicator: potassium chromate. The ashes of saliva contain, according to HAMMERBÄCKER, 18.3 % chlorine.

The presence of chlorine will at first directly increase the osmotic strength in a high degree, as chlorine has a low molecular weight. Chlorine seems, however, to be able

---

[1] ref. HÖBER, 1911. p. 29.

[2] Free carbonic acid does not exist, only the anhydride, but the expression »carbonic acid» has ben generalized.

able to penetrate many kinds of tissues — though probably not carious matter. Moreover it also seems to be a poison to the human body in general, although the symptoms are not yet clear. Medical science has of late fixed its attention upon this matter, and a new generation will surely hear such words as: »Don't take more salt than necessary.»

»Salt», we said, because the greatest chlorine-producer we »civilized» people consume is common salt, which in solution dissociates and gives off free chlorine. NaCl dissociates up to 82 %, thus, more than any other salt. Chlorine can further be transported direct into the mouths of workmen in certain industries.

When for a long period the body obtains strongly salted food without sufficient water and fresh food, a special stomatitis appears which is known under the name of scurvy. This is probably a pure salt-stomatitis. Whether the chlorine, or the sodium, or, which is most probable, both of them, form the active part is not yet clear. Its cause and symptoms are quite like the »metal-stomatitis» caused by mercury and lead.

The importance of chlorine lies further in its function as a component of hydrochloric acid (see below).

Ammonia.

The presence of ammonia gas is shown by titration with HCl solution.

Ammonia is a product of fermentation of certain animal food and animal tissues in general. Thus the saliva itself produces by fermentation of microorganisms alkaline products. Its *sine qua non* is consequently bad hygiene. As it very readily forms salts, ammonia is indirectly able to increase the osmotic strength of saliva, having probably the same property as alcohol in regard to permeability [1]. Ammonia is assumably the most com-

[1] s. Bünte u. Moral, D. M. f. Z. 2. 1910. 81.

mon, foreign (non-natural) alkaline substance in the saliva and consequently the substance upon which the alkaline-theory is based.

Hydrogen.

Hydrogen is probably always present in the mouth in higher or lower concentration. It can be detected by titration of a mixture of acetic acid plus sodium-acetate. Indicator: methyl-violet or benzol-azo-anilin (s. HÖBER [1]). Exact quantitative analysis with help of the »gas-chain method».

Hydrogen in the mouth presumably comes from the organic acids, which, by dissociation, give off free hydrogen. The higher the valency of the acid, the more hydrogen-ions are given off. The importance of hydrogen lies in its affinity to chlorine, resulting in the formation of the harmful acid, hydrochloric acid, the principal mineral acid which appears in the mouth. On account of its low concentration this acid is perhaps not so important from an osmotic point of view, but it is highly probable that it plays an important rôle in the physical processes in the roots of teeth on account of its chemical power of dissolving animal membranes (the root cement), thereby opening the way to physical processes through the teeth. Experiments have shown that hydrochloric acid is able to dissolve the carious matter, although the latter possesses a great power of resistance against all solvents.

Hydrochloric acid is, finally, a catalyst, *i. e.*, here, acid-former, without losing anything in volume. Its catalysing power is up to 400 times greater than that of any organic acid. The presence of this acid can be quantitatively detected by »ester-catalysis», of which more in a later chapter. The presence of neutral salts, *e. g.*, common salt, increases the catalytic power of HCl.

---

[1] HÖBER, 1911 p. 159.

Metal Gases.

As to the »metal gases», their presence in the mouth is, of course, of a more temporary nature and is associated with industrial work, medical treatments, accidents, poison, and such like. When present in large quantities and for a long time, the saliva, saturated with the gases in discussion, causes stomatitis, which we may call »metal-stomatitis», the well-known symptoms of which are quite similar to one another and to the salt-stomatitis or scurvy previously mentioned. It seems to be associated more with the changed composition of saliva than with the special pathogenic action or corrosive properties of the gases. So here there is possibly an osmotic pathological case. At any rate this »metal-stomatitis», as well as scurvy, favours crown-caries in early years and root-caries with a certain success in older years. A stomatitis lasting a long time will annihilate the gingiva and cause a retraction round the neck of the teeth of several millimetres. The consequences in the form of open caries or loosening teeth will be seen only long afterwards.

The »metal-gases» (and metaloid-gases) to which we refer are phosphorus, mercury, lead, antimony, arsenic, sulphur, etc.

The presence in the mouth of dentures with metal clasps, and all other artificial dental arrangements of metals certainly effect indirectly the formation of salts and acids, especially mineral acids (viz., HCl, HNO_3).

The Saliva and the Hydrochloric-Acid from the Gastric Juice.

The previously-mentioned property of HCl being a strong catalytic agent has a great importance as regards the rising of gastric juice into the mouth. This phenomenon, which is a consequence of too great acidity in the stomach, depends for its occurrence upon a surplus of the

chlorine (*e. g.* from NaCl) and free H-ions (*e. g.* from organic acids or other substances producing free hydrogen). The more salt in the stomach, where free hydrogen is always at hand, the more hydrochloric acid in the gastric juice, and consequently in the mouth. »Das funktionell Wichtige für den Magensaft», MICHAELIS writes [1], »ist allein die Wasserstoff-ionen-concentration [= (H.)] und, wenn man so will, kann man die (H.) auch als »freie Salzsäure» bezeichnen; den diejenigen negativen Ionen, welche als Gegenpart der freien H-ionen vorhanden sind, sind ja Cl-ionen». The quantity of free HCl in the gastric juice can vary, according to HÖBER [2], between 2—9 milligrammes per one gm. of the gastric liquid [3]. Certainly the lower figure should be the normal one. The surplus quantity in cases of high acidity, however, which the alkaline liquids, including the swallowed saliva, cannot neutralize or reduce, will be moved by the stomach deliberately upwards, and thus hydrochloric acid appears in the mouth.

Here the harmful acid operates at first as a catalyst, successively increasing the quantity of acids. People who have too high an acidity of the gastric liquid have thus, solely on account of this circumstance, at certain longer or shorter intervals a saliva that is chemically and physically very aggressive.

The way in which the hydrochloric acid originates in the body is still unknown. For his own humble part the author is inclined to assume that HCl is a *secondary* product of the sodium chloride in the gastric juice, and that it cannot be primarily formed by the human body itself or its glands. Thus: the first grain of NaCl in the stomach will by transformation and dissociation produce the first drop of HCl, and when for a time present, rare pathological states only are able to drive it quite away (cf. MICHAELIS, l. c. chapter 50).

---

[1] MICHAELIS, 1914 p. 111.

[2] HÖBER, 1911 p. 180.

[3] S. also MICHAELIS, l. c. p. 110.

The Secretion Salts.

The inorganic salts pass through the body and are eliminated by milk, sweat, urine, saliva, etc. »Sodium chloride as well as potassium chloride leaves the body in just the same form as it enters it, and thus passes through the body unchanged.» [1] This, however, does not mean that these salts are not necessary to the body.

A part of the salts introduced is separated by the saliva, and examples have been given earlier in this work showing how the injection of common salt in the jugular vein of a dog is followed by an increased quantity of chlorine (the anion of common salt). Consequently, the more inorganic salts that the body gets the more salts will the saliva separate. As regards Ca, however, cf. test by Bunting & Wixon [2], according to which an increase of calcium in the food had no effect at all upon the rate of CaO in the saliva. The inorganic salts partly pass through the mouth twice, which thus increases the osmotic strength of the latter, and no mouth hygiene can hinder the oral liquid from being a strong osmotic agent in the night.

Most of the inorganic salts operate in the same way, and the result thus depends upon the quantity introduced. Of course, chemical compounds can appear and in that way alter the result. Sugar, on the contrary, is harmful to the teeth when passing through the mouth only, and has probably no influence upon the secretion of the salivary gland. If, for instance, one side of the jaws is inactive in mastication, we immediately see what large layers of »tartar» are built up from the secretion salts.

Marshall's very careful researches of the constitution of saliva (published 1915, 1916 a. 1917) show that the saliva in carious mouths *has a relatively higher amount of inorganic salts* than the saliva from immune mouths [3].

[1] Höber, 1911 p. 385. cf. also Meyer & Gottlieb, 1914 p. 545.

[2] Bunting & Wixon, The Journal of Internat. Dent. Ass. 1917. 81.

[3] Marshall, The American Journal of Physiology 1917 p. 221.

It is evident that not only the pure presence of secretion salt renders the saliva harmful, but the concentration of the salts as well plays a decisive rôle. The concentration depends, however, not only upon the absolute quantity of salt, but also upon the relative. If thus the body at the same time gets plenty of salts and plenty of fresh common water (not carbonic-acidulated or mineral-water), the danger will be evaded. This applies, of course, to all kinds of harmful materials.

Mouth-breathers who have a dry mouth with very little, and, we may add, very concentrated saliva, have generally decayed teeth.

Summary.

1. It stands as a fact both empirically and experimentally that the saliva during certain intervals obtains osmotic superpressure to blood.

2. The saliva obtains its osmotic surplus by means of chemical materials, especially sodium chloride, cane sugar and medical salts.

3. Saliva having an osmotic superpressure tends to press out the blood through the membranes.

Criticism.

In this chapter the saliva is treated from points of view which concern the possibility of finding higher values for the osmotic pressure of the saliva than those which are known from the official figures. In spite of the fact that the figures obtained are in opposition to the official ones, found in the text-books, they nevertheless furnish a conclusive proof as to the question in discussion.

The osmotic division of the liquids of the human body into »constant» and »inconstant» ones is, of course, not correct in an *absolute* sense.

Titration is, finally, no exact method of analysis. Objection may be raised against the use of methyl-orange as saliva-indicator.

CHAPTER VI.

Subjective or Constitutional Factors of Primary Caries.

Introduction.

If for prosthetic or other purposes we trepane a caries-free tooth and extirpate the pulp, leaving the canal thus formed unfilled, the tooth will soon be annihilated, even if the mouth has otherwise no caries, and we shall find, on investigating the centre of the tooth, that the dentine has become decomposed. But the process is generally different from the usual. The dentine is grey and soft, (exceptions of course in the case of smokers); not brown-black in colour and tough as is otherwise the case. What we find is undoubtedly the result of a purely chemical microorganic annihilation process, working quite free from the influence of the pulp. The *sine qua non* for this chemical annihilation process is, of course, the entrance to the inside of the tooth. But here we have artificially opened a way. The main question will now be: What circumstance makes it possible in natural cases for the chemical annihilation process to enter through the wall of the tooth?

The common opinion is, as known, that acids — formed by decomposition of carbohydrates — are able to open the doors for this destructive process. That in rare cases acids — produced in different ways — really can melt down the protecting membrane is correct, but in abnormal cases only, *viz.*, at the front of teeth of very weak children in case of an excessively bad oral state — in temporary as well as permanent teeth — and in cases of half-erupted

teeth (*e. g.*, wisdom teeth) lying in a pocket of the gum, or artificially on account of an operative injury or different kinds of dentures. This external destructive acid attack, »white decay», does not necessarily lead to »caries,» whether chronic or open; and further, this chemical erosion has a distinctly pronounced character with its white colour and its *irregular* contours spreading far on the surface.

Notwithstanding this our investigators have reasoned as follows: Here we see that acids are able to open entrances leading to caries, what is than more natural than that all caries is based upon acids as openers of entrances? They have generalized from the single case and in that way built up the chemico-parasitic theory. But then comes the fact that the appearance of common caries, especially at an early stage, stands in striking opposition to the superficial erosions, which we assumed to be of acid origin. In order to evade this pitfall the Millerites have invented the convenient ideas of *loci minoris resistentiae* and acids operating in *statu nascendi*, excellent latin expressions to explain what none can positively deny. But, it is easy to prove *negatively* that these pretentions are built upon a very weak ground. Plenty of empirically known facts speak against them, but here we need cite three only:

Firstly, that teeth with deep congenital defects (open entrances to dentine) very often, indeed, as a rule, are free from (open) caries, where caries is to be found elsewhere in the same mouth. »The pits were unusually deep and sharp forming what seemed to be unusually favorable places for caries to begin, yet the enamel was perfect in the bottom of all of these pits.» (Black); [1]

Secondly, that we often see mouths with the front teeth carious, but the *fissures* of premolars and molars intact; and,

Thirdly, that it can be experimentally shown by

---

[1] Black 1908 I p. 125.

anybody that all diluted organic acids have very little superficial chemical influence on the fully-built teeth *in vitro* and consequently still less *in vivo* (on account of the neutralizing effect of saliva).[1]

The fact that a longitudinal transverse section, cut through even the smallest carious area and the pulp shows (if correctly made) a macroscopically visible, coloured connection, containing iron and blood, between these two points, is a circumstance which must lead every thinking odontologist to the opinion that the initial attack upon a tooth is the result of a reciprocal action and must have an objective and subjective cause, for which claim the symmetrical appearance of caries among many other points of interest also speaks; a purely objective agent could not possibly develop a special »desire» for certain forms of teeth.

We are, thus, obliged to assert an originative, or primary state of caries and a consecutive or secondary, *viz.*, the chemico-bacteriological annihilation process. The latter follows upon the action of the primary caries or after a tooth, by trauma or by dental arrangements, has suffered an entrance to its inside. (See further »Secondary Caries».)

The differences between primary and secondary caries may be regarded as follows:

Primary Caries:

Is an osmotic-pathological process.

It is deep caries and grows eccentrically from the pulp.

It destroys the enamel by undermining.

It is commonly reddish-yellow to dark brown.

It is characterized by a reddish or yellow-brown or brown-black spot on the enamel or the root, and a coloured conic zone in the dentine, forming a macroscopically

---

[1] cf. Brugnatelli, 1913 p. 254.

visible connection with the pulp, and contains blood-matter.

It is not necessarily accompanied by a loss of dental substance.

Secondary Caries:

Is a chemical-bacteriological process.

It is superficial and grows concentrically to the pulp.

It destroys the enamel by erosion.

It is, when operating independently of primary caries (»artificial caries»), white grey to yellow grey to salmon red(?).

It always involves loss of dental substance.

It may be remarked, firstly, that this distinction is *genetical* and that in reality the primary and secondary caries in most cases of open caries merge together in the clinical carious state; secondly, that the objective reasons of these definitions will be given in the following; thirdly, that if man did *not* operate in the human mouth with fillings, appliances, dentures etc., secondary caries would be a pure consequence of primary caries, the latter being *sine qua non* to the former.[1] In order to be able to understand the problem of caries, however, it is necessary to know the difference between these two stages.

We are now going to treat the subjective and objective factors which generate primary and secondary caries, beginning with the former. By subjective factors we mean the internal circumstances or the pure constitutional-dental facts; by objective or salivary[2] factors the external circumstances, *i. e.*, the conditions of the mouth, the food and saliva, which in conjunction with the subjective factors produce caries.

---

[1] Except in the case of deep traumatic injuries only.

[2] Not to be confused with the similar expression of MARSHALL.

Subjective Factors of Primary Caries.

These are: —

1. Point or area of attack: defect of enamel or absence of enamel. These defects can be of a histological, a structural, a traumatic or a chemical nature. There is no doubt that the histological entrances are the most frequent. This claim is confirmed empirically as well as experimentally. The difference between histological and structural defects is only one of size. The former are microscopical or point-like entrances. The latter are to be found in the fissures, in the cingula, proximally, etc., or consist of pits, congenital hypoplasia, Hutchinson's defects, etc.

If caries arises in traumatic defects these must be very deep. It is especially the neck that is susceptible to trauma. Even very rough knocks on the crown of a tooth do not lead to caries, which our daily experience verifies.

Carious entrances of a chemical nature are also very rare as regards crown caries and occur in special cases only; labially on young teeth and in slow erupted wisdom teeth and, of course, through application of dentures.

The expression »absence of enamel» refers to the root of the tooth. Here strong acidity may be able to corrode the outer membrane of the root and in that way *favour* the action of primary caries arising.

2. Convergence of dentine-tubules from the preliminary defect of the pulp. The tubules form the path between the enamel and the pulp. If the latter is so far contracted that the tubules from the point of caries do not reach the pulp, the carious point will stop as chronic caries, a fact which we daily observe. The new-dentine will probably also render difficult, if it does not mechanically quite stop, any connection between the point of attack and the pulp. [1]

[1] Kantorwics, D. M. f. Z. 8. 1910. 559.

Those dentine-tubules that are involved in the carious process and which can be macroscopically seen in specially and correctly ground sections of carious teeth as a yellow-red band from pulp to caries, have been called the caries canals by the author, as stated above. In order to understand the justification of this name — perhaps a better can be found — we must bear in mind that in a section this »canal» is a coloured zone, while *intra vitam* it is a conic group of tubules, through which plasm is flowing.

3. Living pulp. That a living pulp must be the *sine qua non* to a process which is giving off a flow of plasm from the pulp chamber up to the point of attack is clear. The younger the tooth the larger and more vigorous is the pulp, and the chances of connection with saliva are correspondingly greater. (s. further Chapter X).

It has already been shown in a previous chapter that caries and pulp stand in direct relationship, so that the contraction of the pulp also diminishes the possibility of crown caries arising.

We may divide primary caries into two different kinds with regard to the entrance, namely, *crown caries*, passing through the enamel, and *neck* or rather *root caries*, entering below the border of enamel. On account of the histological fact that the neck part of the enamel is often very thin, and therefore porous and less resistant, we could in fact speak of a third class of caries, *i. e.*, in the border of the enamel, but in practice we are justified in reckoning with crown and root caries only. To the latter kind we then reckon the singular defects which are known as gingival erosions.

The simplest or the first form of primary caries is a yellow or lightred-brown area on the enamel, or, perhaps rather, in an anatomic depression (defect) of it — micro or macroscopical, which depression is connected with the living pulp by a very thin canal. If a dental

region, *e. g.*, the front teeth, has more or less caries, all its histological defects, *e. g.*, erosions, will most often show primary caries. If, further, a certain tooth has open caries, most often all its erosions also have a caries-canal to the pulp. Primary caries on the root is characterized as a yellow-reddish colouring.

Crown Caries.

Because the *fully-built* enamel is impermeable *in vivo* to physical and, practically, insensitive to chemical influence except in the case of artificial prosthetic arrangements, which is shown, partly by the experiments, partly deduced empirically [1], it follows, that natural [2] crown caries, when acquired, *must arise before this state is entered.* Crown caries in teeth having almost fully built enamel on eruption is consequently established in the earliest months (weeks) of the life of such a tooth (after eruption).

In teeth having not fully organized enamel by eruption (mainly on account of too early eruption), the time of establishing crown caries coincides with that time, which elapses until the enamel has reached full organization. Crown caries in such teeth is consequently established in the earliest years (months) after eruption.

As regards the different places where crown caries appears, we can, further, speak of: —

1. Fissure caries and caries in the cingula,
2. Labial caries and
3. Proximal caries.

Places where caries never or practically never enters are the lingual crown sides of premolars and inferior front teeth. The cause of this must be ascribed to the total absence of histological folds or porosities on these surfaces, which is, further, derived from the fact that the lingual

[1] cf., however, Bunting & Rickert, The Journal of Nat. Dent. Ass. 5. 1918. 519 with Chapt. X.

[2] = dental appliances not present.

sides of the teeth in general seem to escape caries more than any other. The cause is probably also ascribable to favourable circumstances with regard to the connection with the contracting pulp, *viz.*, the direction of the lingual dentine-tubules, and to the length and the thickness of the lingual enamel part, *i. e.*, favourable histological factors, (cf. TÜRKHEIM [1] and PICKERILL [2]) and,finally, also to the fact that the lingual part of the teeth is for rather a long time concealed by the gingiva.

That caries prefers the *fissure* and *cingulum* does not need any explanation, for these entrances are even macroscopically to be regarded as histological entrances to the inner part of the tooth. In the fissures of molars and premolars as well as in foramina coeca of upper incisors, the enamel shows a very incomplete structure so that those places form *loci min. resistentiae.* These circumstances have also been used by the advocates of MILLER, who holds that freedom from disturbance is the *sole* condition for caries. They have also been used to explain the fact that the lingual sides are generally free from caries. However, the presence alone of labial caries is enough to change the position of the matter.

Labial caries has different forms of development. It appears as gingival caries, or it spreads itself over the whole labial surface. By one author, FEILER [3], it has been called »Circular Caries» when appearing in children's teeth, which form is probably developed from common labial caries in the case of very resistless structure of the tooth. This kind of caries seems almost exclusively to be found in resorption-teeth (deceduous teeth) of marasmic children. It grows at a terrible speed and devastates the teeth shortly after eruption. *As the last remains of the tooth only the lingual side and the labial edge of the crown are to be seen.* The early or common form of labial caries,

[1] TÜRKHEIM, D. M. f. Z. 10. 1914. 746.
[2] PICKERILL, Dental Cosmos, 10. 1913. 969.
[3] WITZEL, Deutsche Zahnheilkunde 29. 1913.

which is called gingival caries, has, as is known, quite a special oval form, according to its connection with the pulp and the dentine-tubules. Does anyone think that a pure chemical process would always show such a specific form?

In Chapter I we have shown that the most susceptible part of the crown is the neck part, where the state of completion is first reached in a more advanced age of the tooth. The most susceptible region of the crown is thus forming a zone round the tooth (gingiva not retracted) limited by the gum and the papilla, where the histological porosities are numerous, and consequently, in this belt of enamel, which of course, also comprises the proximal surfaces, the possibility of caries arising is rather great.

It will otherwise be difficult to explain the frequent presence of *proximal caries,* or perhaps we may say proximal crown caries, for, as we shall soon see, there are two kinds of such caries. That it stands in connection with the fact that physical or chemical processes require freedom from disturbance seems almost sure, but this circumstance is not at all sufficient to explain the frequency of proximal caries. Cf the following lines by Black [1] concerning a case of proximal caries: »We might ask here, by what power, circumstance or condition has the action of the acid been confined to this one small spot, while all the rest of the surface of the tooth is free from any action of the acid?» The facts prove that teeth which are very spread out nevertheless acquire proximal caries. Circumstances which contribute to the frequency of proximal caries are presumably the following:

1. The enamel is, as a rule, shorter (reckoned from the cusp) proximally, than lingually and buccally. The enamel at eruption is weaker and thinner (more porous) in those parts which adjoin the root than in the parts above. Consequently the weakest (histologically most

[1] Black, 1908 I p. 77.

incomplete) part of the enamel proximally lies »higher», *i. e.*, nearer the cusp, than buccally and lingually.

2. The distance from the pulp-wall to the proximal surface is in a newly-erupted tooth comparatively shorter.

3. A tooth, newly erupted, has a weak, porous enamel here and there, with more numerous porosities in certain regions. If harmful agents are in action, they tend to attack all weak points of the enamel, but the parts (or points) which are rubbed by the lips and tongue and further hardened by the saliva (if saliva has, as HEAD [1] states, this property), will be — as all physical and chemical processes need freedom from disturbance — free from open caries and the enamel here successively stronger and impermeable. But the points, however, which are not favoured in that way, *i, e.*, the proximal surfaces and the fissures, will acquire caries.

The Symmetrical Appearance of Crown Caries.

As all dentists know, caries sometimes appears not only symmetrically as regards kinds of teeth but also as regards the place of the teeth. The author thus observed in a mouth, otherwise quite free from caries, that both the upper first molars had a small carious cavern at the mesio-lingual corner, which cavities had evidently arisen in histological defects.

These phenomena completely exclude a chemico-parasitic theory as genesis of dental caries which *eo ipso* is operating quite planless. Can they be explained by an osmotic theory?

According to this theory crown caries (root caries does not seem to appear symmetrically) is developed immediately after the tooth is cut, if harmful agents are at hand. But the teeth erupt in groups, *i. e.*, the front

---

[1] HEAD, Dental Cosmos 1907. 802. cf MILLER, 1889 pp. 157 and 171.

teeth at a special time, then the premolars, then the canine teeth, etc. If during this special eruption time the individual gets a rather abundant supply of sugar in different forms, medical preparations or highly salted food, than both before or afterwards, the consequence will be that just these teeth will be attacked, but no others.

The characteristic case when the two upper canine teeth, the strongest of the bite, are carious, but the other teeth in the mouth intact, in that way obtains its simple ground of explanation.

The existence of symmetrically appearing caries is one of the most pregnant facts in the systematic of caries.

The Causes of Latent Crown Caries Becoming Active.

The appearance of chronic or latent caries is well known. We daily see deep-brown plaques on the proximal sides of teeth, as well as in the fissures or in other places, which coloured zones have been standing unchanged for years. The reasons of these facts are given in chapter I, but as we also know, that such chronic caries will sometimes again become active (= Transformation into secondary caries.)

The causes of this transformation from latent into open caries may be divided into subjective and objective ones.

A. Subjective Cause: the Death or Irritation of the Pulp.

1. A root-filling of the tooth. If a tooth has chronic caries distally, but open caries mesially and the tooth is root-filled from the mesial cavity, the former, the distal cavity, will most often recommence, assumably on account of the tooth having lost its means of protection, *i. e.*, the pulp, which before held its own against the distal chronic caries canal. Caries which appears in a tooth after a root-filling has been made is always identical with resuscitated

latent caries, unless an entrance is formed in an artificial (not natural) way, *e. g.*, by dentures.

2. A simple filling (pulp living), *e. g.*, in the occlusal surface of a tooth, will often irritate and render the pulp weak and in that way or another awaken a slumbering cavity in another place of the tooth. This is a general experience.

3. At a certain later period gingival primary caries is most often at hand, even if life (pulp) keeps the process inactive (not open). If the tooth, however, is extracted and put in a mouth as an artificial crown the process will swiftly proceed.

B. **Objective Causes of Recommencing Latent Primary Caries.**

a. Local objective causes.

1. Trauma of the gingival border of the crown of a tooth, on account of grinding for fitting an artificial crown on the adjacent tooth.

2. A metal filling in the adjoining tooth, the filling giving off material which, dissolved in saliva, increases the local osmotic pressure at the proximal side, produces acids or operates like a catalyst or by unsuitable construction forms a retention-place, which results in inflammation leading to a contraction of the papilla.

3. A gold crown will often cause open caries in the neighbouring tooth, which may certainly be sometimes explained as a consequence of the grinding, which has damaged the sensitive gingival part of the neighbouring tooth or irritated the papilla, but it can also be, and probably most often is to be, ascribed to dissolved metal products in saliva, which here, quite as in the case of metalfilling, at the proximal side, can locally supply an osmotically stronger liquid or contribute to a formation of mineral acids.

4. Dental appliances of metal or vulcanite and all

other circumstances which produce a local state of increased physical and chemical action.

b. General objective causes.

1. That diseases sometimes cause a destruction of the teeth we know empirically, but we also know that in other cases people succumb to, *e. g.*, tuberculosis, and have excellent teeth. On account of what is written above this is nothing remarkable. In the former case it is a question of resuscitating latent caries, already present, but in teeth which are free from such, or have their pulps very much contracted, no diseases at all, as well as no harmful agents in general, will be able to damage the dental tissues.

Maladies known to cause a decrease of the osmotic pressure are, *e. g.*, typhus, certain forms of nephritis and neurasthenia, but certainly many others. Diseases which result in an increase of the pressure are, *e. g.*, malaria, certain forms of nephritis, congenital cyanosis, apoplexy and certain cardiacal maladies. It is obvious that the maladies which diminish the osmotic pressure of the blood will osmotically result in a weakening of the pulp-liquid in relationship to the mouth-liquid. (s. Eckermann [1]).

For the rest, the time at which the diseases originate is of great importance, the time during and after eruption being decisive.

2. The empirical circumstance that the state of pregnancy often has a bad influence is confirmed by the fact that in all languages there is a saying to the effect that each child involves the loss of a tooth. That the time of pregnancy sometimes produces a sinking of the osmotic pressure is scientifically proven (Mohr and Stehelin [2]). The cause of it is probably that in weak women having bad regeneration-power, the foetus takes up too swiftly the salts of the blood, which cannot quite

---

[1] Eckermann, Sv. Tandl. T. 4. 1913. 380.

[2] Mohr und Stehelin, 1912 p. 113.

so swiftly be restored. (Compare the desire pregnant women sometimes have for eating mineral substances, ashes, etc.). The lower the osmotic strength of the pulp-blood, the easier it will be for the saliva to acquire superpressure. Just the latent caries will then be influenced, which we are able to understand as we empirically know that it is the teeth, that are already carious or filled which swiftly succumb to caries.

It should also be said that the state of pregnancy is often to be regarded as a weakened condition analogous to constitutional maladies involving a lessening of the resistance of the tooth and resulting in a transformation of primary chronic caries into secondary.

That much discussed case — the inexplicable causality between caries and pregnancy — obtains in this manner a natural and simple explanation. Of course, unsound states of ventriculus and nephritis are contributing circumstances. [1]

3. Another general objective cause favouring a recommencement of latent caries is a considerably increased acidity of saliva, the acidose being produced principally by mineral acids (hydrochloric acid) as well as an augmentation of all those factors that in general cause primary caries.

Root Caries.

The tooth is normally immune to crown caries five — fifteen years after eruption — provided that meantime no caries has appeared — the shorter or longer time depends upon the mastication pressure developed (cf Chapter I). As seen, we speak here only about caries in permanent teeth. The conditions in temporary teeth are analogous, but within narrower limits of time. But now

[1] cf. *inter alia* Michaelis, 1917 with
Liesegang, D. M. f. Z. 5. 1915. 187,
Grüner, Deutsche Zahnheilkunde 34. 1915 and
Kunert, D. M. f. Z. 5. 1915. 157.

the tooth enters a new period of danger — the time of gingival caries, neck caries or root caries, which latter name is the most correct. Within certain limitations we are right in asserting that this period lasts from the time when the possibility of acquiring crown caries has disappeared to the end of life.

Thus, time for crown caries, from eruption to about the 30th year; time for root caries, from the 30th year to death.

The character of the boundary between crown and root, between enamel and cement, is, however, very different in different individuals. [1]

Attention may here be directed to two points.

1. Crown caries can arise after this time, but it is then a question of resuscitating latent caries; and

2. Proximal caries after the 30th year originally is most often root and not recommencing crown caries.

If the enamel is the means of protection for the crown, so also is the gingiva the protecting membrane for the root and for a certain time also for young enamel round the neck. If it is necessary for the carious process to get an entrance through the enamel in order to enter the inside of the tooth, it will, of course, be very easy to penetrate the dental tissues at the exposed neck. And we can say that the exposed neck of the tooth will, in practice, nearly always involve root caries, more or less, in the form of primary caries, if caries is to be found in the mouth in question. The difference between primary and secondary root-caries is, as said the difference between closed and open caries. Primary caries is to be seen as a colouration, more or less red, at the exposed neck. Secondary caries means that the process has changed into loss of substance. According to the chemico-parasitic theory an exposed root should be a swift spoil to caries,

[1] cf. Thorsen, Skand. Tandl. För. Halfsekelsjub. 1916.

but in fact the change into open caries goes on very slowly and can linger for a long time as latent root caries. A recommencement is dependent upon the same factors as those which cause recurrence of crown caries in general, above all, upon the death of the pulp.

The early form of crown caries through the enamel has — in any case at the beginning of the process — a round form, labially an oval form, but initially the entrance of root caries often has a spreading irregular form, following the limit of the enamel and depending upon the size of the exposed part of the neck.

When penetrating deeper in a more advanced state and when occurring labially, root caries, however, acquires a special form, an oval-like form, of which we will speak later on. It is evident that a special, always repeated form of caries cannot be ascribable to agents operating from the outside only, but expressively indicates a reciprocal action between the tooth and the effective agent.

We may once again remind ourselves that the enamel forms a jacket over the crown, which is longer buccally and lingually, but shorter proximally, Here — proximally — the enamel forms as a rule an angle or arch, which is normally protected by the papilla. We may say without exaggerating that this angle area is that space of the tooth which is most exposed to caries. As soon as the the papilla is damaged and retracted, this angle will be revealed and caries appears, if the objective agent is in function and if the contraction of the pulp is not very much advanced. This kind of caries is also »proximal caries,» but, genetically, of another kind than proximal crown caries. »Fresh» proximal caries in people over fifty years of age has always started as root caries.

The Causes of Receding Gingiva.

We must ask ourselves what the causes of recession of the gum — this eminently caries-favouring factor — may be, and get as answer:

1. Repeated tests and experiments performed for quite another purpose proved that common salt resorbs the gingiva. It is evident, however, that in young and healthy people with very active glands, producing alkaline liquid in abundance, the effect of the common salt must be rather small (cf. Chapter V). Alkaline, we said, because it is not, or probably not, the neutral salt itself which is the harmful agent, but its product of dissociation — the hydrochloric acid (cf Chapter IX) — in any case the chlorine-ions.

2. HCl in too rich a quantity coming from the gastric juice. As the HCl is a strong catalyst, it will then even in small quantities produce high acidity.

3. All brushing with strong chemical preparations, acids, alkalies and electrolytes must in the long run have a deleterious effect upon the gingiva.

4. Dental appliances of different kinds — of metal or rubber which serve as retention-pockets for food and in that way indirectly produce chemical agents (mineral acids?) which »burn up» the gingiva — by producing at first an inflammation, then successively afterwards an atrophy.

5. Trauma, *e. g.*, indiscreet use of tooth-picks, the necessary use of which is commonly a consequence of malpositions, which in its order is often a consequence of extractions.

6. Stomatitis, pharyngitis and laryngitis generated by pathological agents, chemical poisons or by excessive use of alcohol or tobacco.

7. Sedimentation of tartar on account of inefficient mastication.

Gingival Erosions — a Special Form of Root Caries.

The most singular type of caries (= loss of dental substance) is the gingival erosion, also called the »wedge-formed defect». This form of caries is, according to the chemico-parasitic theory, simply inexplicable. How would the process go on, if it was caused by acids according to MILLER,[1] by fermentative carbo-hydrates according to WALLACE,[2] by sugar according to COLYER[3] (caries present in the mouth in all cases)? As soon as the neck had lost its protecting gingiva the destructive process would go on round the neck under quite indefinite mathematical forms, and quickly result in the crown falling off.

Let us see how the matter stands in the light of the osmotic theory. The osmotic caries-process is dependent upon three trigonometrical facts: —

1. The width of the pulp-chamber (and the pulp).
2. The course and direction of the dentine-tubules.
3. An external operation-base; either an histological defect in the enamel or more or less unprotected root.

If we make a section through a gingival erosion, we immediately see how just these mathematical conditions fulfil the idea of this type of caries. We can leave aside the root-proximal caries, which process is analogous to crown-proximal caries and only think of the labial forms of root caries. We then know that even the advanced gingival crown caries has quite a typical form — oval defect with pointed ends. This form is also characteristic of the labial root caries, and its special form the gingival erosions. The latter differs considerably from common caries to the superficial observer. It is without carious matter and has very smooth walls.

This circumstance depends, however, upon the direction of the osmosis. The common situation is that

---

[1] MILLER, 1889.
[2] WALLACE, 1914.
[3] COLYER, Dental Record 1; 12. 1916.

the saliva is the osmotically stronger liquid, *i. e.*, the diosmosis goes *from* pulp outwardly, but here the case is the opposite; the pulp is able to arrange a diosmosis in the direction of the pulp, probably after powerful acidity has corroded the external membrane of the root. On account of this, the tubuli of the dental tissue are expanded and the intertubular substance destroyed is transported away and the erosion takes the form of a truncated cone, the top being equal to the width of the pulp-chamber and the side wall being the tubules which are starting from the active part of the pulp; the apical rim of the erosion is formed by the curve of the gingiva (at the time the erosion was commenced) and, finally, the cuspidal rim is the labial rim of the enamel, or, if the pulp is deeply contracted, the cuspidal roof of the pulp-chamber. (s. further Ch. X).

The gingival erosion can thus be defined as a form of root caries lying quite free from the enamel. On account of the absence of the protecting carious matter, which in its order is a consequence of the direction of the osmosis, substances which are able to penetrate the dentine (saliva with a rel. high rate of mineral-acids) will also possibly enter through the erosion into the pulp.

The suspicion that the gingival erosions were a result of too energetic brushing is contradicted by the fact that plenty of skulls from the Middle Ages show these defects.

As regard further opinions of the etiology of the gingival erosious, see the detailed criticism by Klöser.[1]

We cite the following lines by Cohn[2]: »I had touched the exposed root of a canine tooth (the gingiva and bone of which had been taken away by me on account of pyorrhoea), with lunar caustic and found after extraction the front part of the root up to the root-canal coloured smoke-gray.»

---

[1] Klöser, D. M. f. Z. 3. 1914. 216.
[2] Cohn, 1900 p. 216.

We can add: up to the root-canal, but not farther, for if here diffusion (= capillary action) was *alone* at work the silver stain would have coloured the greater part of the root and crown grey. And if, for control, a tooth, having had the apex shut up, is put into diluted solution nitr. arg. for some months, we shall find after splitting the tooth that no colour matter at all has entered the tooth (no penetration through the enamel or the dentine of the root.). This test proves that (1) lunar caustic enters the centre of the tooth only under influence of life; (2) »influence of life» must here mean osmosis or diffusion; (3) this communication by *Cohn* is thus a good support of the explanation of gingival erosions.

Summary.

1. In this work we distinguish, genetically, between primary caries, which does not involve loss of dental substance, and secondary caries, which always involves loss of dental substance.

2. Both these stages form together the *clinical* idea of caries.

3. In a normal state secondary caries follows the path formed by primary caries, but in abnormal cases — dental appliances in the mouth — secondary caries alone (= strong mineral-acidulated local fermentation) is able to open an entrance through the tooth, of course, easier through the root than the crown.

4. We must distinguish genetically between (primary) crown caries and root caries. Crown caries is established at time of eruption, root caries in a later period of life. Clinically, advanced root caries obtains the appearance of crown caries.

5. Primary caries is characterized by its power of stagnation (chronic caries), permanently or temporarily.

6. The reinflating of chronic caries is due to an abnormal external state of the mouth, caused by subnormal

appliances (dentures) b) disturbance or the extinguishing of the life of the tooth.

7. Gingival erosions are the product of agents which in cooperating with the pulp liquid diosmose through the tubuli terminating in the exposed part of the root, into the pulp.

Criticism.

The new ideas: primary and secondary [1] caries; latent, crown and root caries, etc. — advanced as far as the author knows for the first time in this work [2] — are no artificial inventions, but based upon clinical and empirical facts and quite independent of any theory of the etiology. The systematic of caries is further treated and developed in Chapter VIII.

If man were not operating in the human mouth, primary and secondary caries would stand in simple causality.

---

[1] in the meaning here advocated.

[2] and in previous publications connected with this work.

CHAPTER VII.

Objective or Salivary Factors of Primary Caries.

Introduction.

I.

The natives of the Arctic regions mainly eat meat and according to the claims of some investigators, these people have on that account teeth free from caries, and certain South-Sea people chiefly eat carbo-hydrates and, therefore, have teeth untouched by caries, from which we may draw the genial conclusion that the »food» itself does not play a positive rôle in the genesis of caries.

»In so far as the relative amounts of protein to carbohydrate in the diet are concerned, the data appear to confirm PICKERILL's (1914) conclusions on this point, namely, that the protein-eating races are as susceptible to dental caries as those whose food is mainly carbohydrate» (MARSHALL [1]).

The food passes the alimentary canal from *vestibulum oris* to *anus* with gradually increasing fermentation, decay and time of stay. How is it, then, possible for the food immediately after entering this canal to be able to destroy, during its rather short stay in the mouth, even if reckoning with food debris, the most resistant tissue that the animal world exhibits, when during the rest of its passage it does not at all damage the tissues forming the walls of the alimentary passage?

---

[1] MARSHALL, The American Journal of Physiology 1917. 225.

In general, the preparation of carbo-hydrate food by civilized people tends to rob the food of its fibrous components in order to make the food less and less difficult to chew. The question is, if this procedure will not finally render teeth, as well as jaw, weak and non-resistant, which would then indirectly favour although not directly generate such a peculiar malady as caries. The case is otherwise with regard to the preparation of animal food, albumen and fat. These food-constituents are more expensive and are more easily decayed than carbo-hydrates, they must, therefore, to a great extent be preserved. The way of preservation is now-a-days different from that formerly employed. Formerly animal food was preserved by drying in the air, now it is preserved by chemical means, *i. e.*, salt and saltpetre, which has made possible an increase in the consumption of meat and fat, and, consequently, in a corresponding degree also of the preservative.

It should also be remembered that an augmented consumption of chemical preparations stands in intimate relationship to the development of »civilization» and »inter-communication» and, further, that great and swift carious devastation in mouths in certain cases may be put into direct relationship with a large consumption of sugar, medical treatment and close contact with certain chemical materials.

Blunschli[1] relates the following concerning caries in wild apes and apes in captivity. He compares the same species in the neighbourhood of Para in S. America. He states that the wild apes never have, (excl. traumatic causes) while the other often have caries. As they drink the same water and live in the same climate, he draws the conclusion, that the cause of caries is due to the food, which for those in captivity is prepared so as to make it easier to chew. He does not think of *sweets*, *sugar* and that the

[1] Blunschli, Schweiz. Vierteljahrschr. f. Zahnheilkunde 3. 1913.

prepared food is seasoned (salted) when given to the apes. Cf.: »Beside bananas the apes get wheat bread and sugar as in all Zoological gardens» (KUNERT [1]).

Reasoning in this way — which may be open to criticism — the author came to think of *salts*. At first a very sceptical attitude was taken up towards the thought that common salt — a substance daily devoured in plenty and valued just on behalf of its preservative properties — could be an effective agent in producing caries. Many years of study passed before the author understood how, when and where common salt, like all chemicals, affect the human teeth.

Further, the following phenomena: that caries always first goes direct to the centre of the tooth; that dead teeth show a remarkable power of resistance; that buried carious teeth are found in the earth, where death has stopped instead of — contrary to expectation — enormously hastening the destructive process [2]; that caries often appears symmetrically in certain groups of teeth; that caries, not too far advanced, shows in its external contour a mathematical regularity; that certain teeth show latent proximal caries, but open caries at the neck — all led the author to the idea that caries must be connected with the function of life, a living factor in the direction of a pulp at active work in the process.

At both these points, »chemical salts» and »active function of the pulp» as reciprocal factors of caries, the author was held up for years without being able to construct a bridge between them, until the thought of osmotic processes suddenly occurred; a thought which, like a flash, casts its light over the obscure problem, illuminating and explaining it.

---

[1] KUNERT, D. M. f. Z. 5. 1915. 172.

[2] MANNING, Dental, Cosmos, 1. 1919. 22.

II.

It is, however, interesting to follow the development of the different opinions of the etiology of caries from MILLER's time until now. According to MILLER [1] carbo-hydrates in different forms and microorganisms of indefinite character and qualities are the chief causes of caries. After this, new theories were formed, constituting more or less modifications of his theory.

It was soon clear to many that carbo-hydrates, as such, could not possibly be the real cause. Then SIM WALLACE [2] among others came forward with his modified opinion: not the real carbo-hydrates, but the soft, sticky preparations of certain vegetables are the culprits. BLACK [3] has directed attention to the presence of a gelatinous membrane as a precipitate from the saliva under the protection of which he alleges the acid-fermentation — principally lactic acid — can proceed without being disturbed. WALKHOFF [4] regards defects of structure on account of a poorness in the nourishing salts in the enamel as forming the entrance of the carious process, and regards bread as the principal cause [5]. Then we have COLYER's [6] observations concerning the food of different negro-tribes in South-Africa, the food of which was always prepared in a sticky, soft form, carefully deprived of its fibrous components. The appearance of caries was, nevertheless, very rare. COLYER's researches, which quite confirm the author's opinion that the carbo-hydrates are neutral towards the annihilation of the teeth may be regarded as a veritable death-blow to the theory of the

---

[1] MILLER, 1889.

[2] WALLACE, 1914.

[3] BLACK, 1908 I.

[4] WALKHOFF, 1913.

[5] cf. »Bröd and Karies» (Bread and Caries), discussion between RYGGE, HIRSCH and ECKERMANN in Norsk Tandl.-Forenings Tidende 4; 6; 8. 1918.

[6] COLYER, Dental Record 1916, 1917.

soft carbo-hydrates. In spite of his own results, COLYER, however, comes to the surprising conclusion that sugar and easily soluble carbo-hydrates produce caries. The original point in his theory is, however, that a capillary power is »holding the films of the solution at the affected sides», an opinion the untenability of which is already proven (p. 10).

As to the question of a one-sided or bilateral carious action, *i. e.*, only objective agents, or subjective (dental) and objective (salivary) factors in reciprocal function, it must be remembered, that theories in the latter direction were formulated very early. Clear brains among our colleagues saw that an objective theory only could never satisfactorily explain the peculiar appearance of caries, *viz.*, the symmetrical appearance; the observed fact that often solely the front teeth in a mouth have open caries, while all the fissures of premolars and molars are quite free, or in any case free from open caries. May this be explained by MILLERS, WALLACE'S or COLYER'S theories? Not at all. If, for instance, COLYER'S theory included the whole of the truth as regards the causation of caries, he would have found negro-mouths either quite free from caries or all teeth more or less devastated. Or, in other words, under his supposition, an objective factor, when present in the mouth, must attack all teeth more or less. Unless COLYER regards the »capillary power» of the inter-dental spaces as functional at the time of eruption only. But if so, the property of capillary action belongs to the teeth and not to the sugar. If this conclusion is accepted the next one, *i. e.*, that the teeth take active part in the carious process, is near at hand.

Coincident with the development and elaboration of the acid theory, odontological history thus supplies the modern theories, the aim of which is to clear up the mysterious subjective factor which, together with the harmful objective factors, might explain the seemingly capricious appearance of caries. »Therefore, there is

something lying over beyond the active growth of micro-organisms and acid formation by them, controlling caries of the teeth» (BLACK)[1].

We have first *»subcalcimus»*[2], then *»ethnic degeneration»*, further, *absence* of the *special salts* of which the modern preparation of corn commonly deprives people: »vitamine»[3]. »Do vitamines exist», asks, however READ[4] and he replies: »it appears that vitamine is as difficult to discover as the philosopher's stone.» (p. 118). Then, a long, not yet, ended fight about *supposed histological defects* in the enamel, which hypothetical defects might explain the fact that certain teeth and groups of teeth are attacked, but others left quite free[5].

RÖSE[6], who made researches into the diffusion of caries in the Swedish counties and who started from the hypothesis that the rate of calcium in the earth is the factor that controls the resistance of the teeth of the population in question, found to his surprise that the inhabitants of the large Baltic island, *Gotland*, which is a pure limestone island, had highly decayed teeth. In order to get around this troublesome fact he simply invented the pretension that on account of endogamy the inhabitants of Gotland were »degenerated». Quite ignoring the question, as to how degeneration is able to generate caries, this supposition only shows his ignorance of the laws of society, which in a natural way regulate »social combustion» in such a manner that country families who are strong, sound and »permanent» give off their surplus to the towns, the families of the latter, on the contrary, after 2—3 generations die and disappear — the swifter the larger and unhealthier the cities are.

---

[1] BLACK, 1908 p. 115.
[2] s. a. o. RÖSE, D. M. f. Z. 1908.
[3] cf. KUNERT, D. M. f. Z. 5. 1915.
[4] READ, Medical Times, ref. Dental Record 3. 1919. 117.
[5] s. RYGGE, Skand. Tandl. Föreningens Halfsekelsjub. 1916.
[6] RÖSE, D. M. f. Z. 1904. 735.

As regards that most debated question of *inheritance* as a contributing factor, it seems to be evident that a powerful body has strong hard jaws out of which the teeth are slowly erupted, and therefore their structure should be well developed; but a weak body has weak jaws, with swiftly erupting teeth (as meeting less resistance), having their external structure at eruption not yet fully built and with porous enamel, because the teeth are erupted at the time when normal embryological work has not yet been completed.

These opinions are mentioned by the by only in order to direct attention to the fact that there has been, in opposition to the orthodox theory, a real but vague feeling *that a purely objective theory never solves the problem of the origin of caries.*

No, first on turning from natural food to food rich in minerals, to crystals, spices, condiments, preservatives, stimulants and medicines, do we come into contact with a power which we sometimes call »osmosis», sometimes »galvanic electricity», but which mysterious and terrible power we, perhaps, better call the »crystalloid power». In combat with this enemy the teeth will be beaten.

In order to be effective the harmful crystals or chemical matters must be dissolved in a liquid. The liquid which is always flowing round the teeth is the saliva, and even if we may assume that many other harmful liquids, *e. g.*, mineral waters, hot sugared tea or coffee are exceptionally consumed in such large quantities and retained for such long intervals as to enable them to do harm, we must, bearing in mind the decisive time of eruption, *in practice* reckon with the saliva as the general or normal conveyor of the crystalloids.

If we then ask ourselves in what way the saliva is made osmotically stronger, we reply:

1. Firstly from substances mixed in the food, and from drinks and medicines, when passing into the mouth.
2. Secondly, by secretion-salts from the saliva glands.

3. Thirdly, by HCl rising from the gastric juice.
4. Fourthly, by absorption of gases.

Points 2, 3 and 4 are treated in a previous chapter as belonging more to the property of saliva.

Solids and Liquids which per os Enter the Body and are Able to Increase the Osmotic Strength of the Saliva.

Introductional Discussion.

The theory here advocated terminates in the assumption that the saliva during certain intervals obtains an osmotic increase in strength, and in that way acquires higher osmotic pressure than that of the blood of the pulp. The osmotic pressure of blood is, according to VIOLA and HAMBURGER etc., 6—7 Atm., and according to HÖBER over 8 Atm.[1]

What substances are then in general, able to give a liquid high osmotic pressure?

All kinds of chemical substances dissolvable in the liquid, which are able to increase the osmotic strength according to their molecular weight[2].

Which of these substances are in this way more harmful than others?

Organic or inorganic salts, which are practically the only substances that need to be discussed.

Which of these are especially consumed by man?

Common salt, sugar, medical preparations of nourishment, etc., and mineral water.

Have any of these, empirically, appeared harmful to the teeth?

Yes, it has been empirically established that, *e. g.*, sugar and certain medical and chemical matters are causes

---

1 See footnote p. 91.

2 Elemental thesis. The total osmotic power of a liquid is not a pure problem of addition, whether in a positive or negative direction.

of caries and destroy the teeth. As to sugar it is ancient empirical knowledge already told in the Bible. Common salt has also been shown to be a very harmful preparation to the mouth, when consumed for a long time and in large quantities, *viz.*, during sea expeditions, etc., even if it is not thought to be directly connected with caries (on account of the slow course of the carious process).

Are these substances the only ones which we suspect as the cause of caries?

Yes.

Has, on the other hand, sugar been able in an artificial way to produce *real* caries in (dead) teeth or sections of teeth by careful tests, when put in renewed saliva from a devastated mouth, the liquid being strengthened by the addition of sugar and the solution made acid by *e. g.*, Acid acet. and kept at 37° C.

No, all such experiments lasting for weeks or months have given a negative result as regards producing carious cavities in dead teeth. Nor have small carious plaques, already at hand on the teeth used in the test in discussion, in the slightest degree changed or developed into open caries.

Has humanity in all times consumed these preparations (sugar, etc.), or first in recent times?

Humanity has in all times, if in small quantities only, consumed such preparations, but first during the last fifty years has the consumption of chemical salts, sugar, etc., increased extensively.

Does the consumption of harmful materials correspond with the increased spread of dental decay?

Yes.

Does the consumption of carbo-hydrates, softened food and the like correspond with the increased spread of caries in the last half-century?

Not at all, rather the contrary.

Conclusion.

Quite apart from the proofs which the author has adduced in support of his condemnation of the modern theories, it is to be concluded from what is written above that humanity really consumes osmotically strong substances; the increased consumption of which corresponds with the enormous spread of caries now-a-days. We further see that among these osmotically strong substances, two kinds, sugar and medical preparations, are in the case of human beings expressively fixed by the strong evidence of experience as factors of caries. On the other hand these substances have never been able to produce caries in tests upon *dead* teeth or sections of teeth, *i. e., in vitro* and are further neutral from a chemical point of view, only indirectly forming acids, *viz.,* sugar, iron, chloride, bromic, salicylic salts etc.

Substances Directly Increasing the Osmotic Pressure of Saliva.

As regards the harmfulness of osmotic substances, the following factors are decisive:

a) The greater or lesser osmotic strength, *i. e.,* absolute osmotic strength.

b) The concentration, *i. e.,* relative osmotic strength.

c) The power to penetrate and enter animal tissues.

d) The quantity in which they are generally consumed.

e) Their solubility in saliva. If not soluble they may practically be omitted as factors of caries, *viz.,* acetyl. salicyl.

The osmotic strength depends upon the molecular weight. The lower this is, the stronger the osmotic pressure [1]. The concentration, however, plays in reality the most important part. Theoretically, common salt seems to be the most harmful on account of its great osmotic

[1] Se footnote page 147.

power in low concentration, but in practice sugar may be regarded as the most common cause of caries, which, of course, does not preclude the fact that other materials may contribute and that in *certain* cases quite other preparations than sugar and salt are responsible.

Osmotic substances which are transported by food, etc., are manifold. They may be divided, however, into food-salts, different kinds of sugar, different kinds of alcohol, medical preparations and different drinks and mineral waters.

Salt and saltpetre belong to the »food-salts», both of which are used as preservatives and seasoning, and, further, all those chemical condiments which belong to the blessings of civilization, as well as artificial spices of different kinds, specially prepared dainties and medical preparations.

Sugar.

The chemical subdivisions of sugar [1] impart very low osmotic strength and seem to have a subordinate value only. Grape-sugar, honey-sugar, etc., are osmotically the strongest of all kinds of sugar. They effect an osmotic pressure twice as strong as cane sugar, maize sugar, etc., in the same strength of solution. As is well known, grape sugar is to be found, not only in grapes, but in many fruits. Grape sugar is also formed by fermentation of starch in the mouth, a circumstance which is often pointed out, but seems to have a rather theoretic importance, because the diastase in the mouth (the chemical splitting of the starch by the saliva) ceases in acid reaction and is effective in the presence of chlorides only, especially sodium chloride. Grape sugar is also formed by treating starch with certain other matters, *e. g.*, sulphuric acid.

Cane sugar is, as said, half as strong, osmotically, as grape sugar in the same concentration. A 10 per cent. cane sugar and a 5 per cent. grape sugar solution is

[1] Burchard ang Inglis, 1912 p. 326.

hypertonic to human blood. A concentrated cane sugar solution (*i. e.*. 2 gm. sugar + 1 gm. water) corresponds to an osmotic pressure of about 132 Atm. at 0° C. (theoretically, in practice probably more [1]) This solution corresponds theoretically to 0.36 gm. NaCl + 1 gm. water. The practical osmotic effect of sugar and NaCl, when introduced in the mouth and dissolved in *saliva*, is shown in Chapter V.

Against the claim that sugar develops caries, the objection has been raised that people in the cane sugar countries are daily chewing young shoots of sugar-canes, without any influence upon the frequency of caries. According to others [2] a chewing and *sucking* of the *ripe* canes takes place. In this connection the following facts may be mentioned:

1. The juice of the *ripe* sugar-cane contains a *maximum* of 22 per cent. sugar; commonly 12—14 per cent. only.

2. The *young* cuttings much less.

3. When sucking or chewing sugar a rich salivation takes place (cf Chapter V) while only a little quantity of the juice is introduced at a time.

4. The chewing of the hard sugar cane cuttings is equivalent to good natural hygiene.

5. According to previous tests (Chapter V) it thus need a quantity of 10—15 grm undiluted ripe sugar cane-juice *at a time* (at each time) *in the mouth* in order to do osmotic harm.

6. It is further conceivable that the juice of sugar cane is to be regarded as a hydrophile colloidal solution having far lower osmotic strength than a corresponding pure water solution of sugar [3].

7. Hydrophile colloidal solutions have in general a low osmotic strength. Neutral salts present in these solutions counteract the osmotic effect [3].

---

[1] cf. Berkeley and Hartley, Proceed. Roy. Soc. 3. 1914. 736.

[2] cf. Johnston, 1882 p. 176.

[3] Höber, 1911 pp. 319, 348, 363 cf., also Chapter V.

8. Conclusion: The chewing of cane sugar, ripe or cuttings, involves the presence in the mouth of a sugar-saliva certainly having a concentration far below the harmful one.

The discovery of a chemical method of extracting crystalline sugar from certain industrially cheap vegetables has made it possible for people to eat sugar to an extent which was formerly unthinkable. The consumption of sugar is in England twice as great per head as in other countries. We feel justified in saying that the successive sinking of the price of sugar corresponds to the increase and spread of caries. It is thus questionable if this discovery is such a blessing of chemical science as it is often regarded. The consumption should in any case be limited to a reasonable quantity.

Sodium Cloride.

One author, FEILER [1], speaks of »chloric-caries», and says: »Caries of workmen whose teeth are attacked by a continuous breathing of chloric-gases. This applies, according to what RITTER says, to chloric-bleachers in paper-mills, workmen in artificial wool industries, calico-printers and others. But in these cases the etiology is clear; everything points out the local cause of the harm; in the case of the confectioners the continuous breathing, and the effect of the powdered sugar, which even scrupulous hygiene, too often lacking, cannot counteract; in that of the chloric-workmen the fastening of the harmful acid on the labial surfaces of the teeth . . . . . *Jurish* mentions amongst others one case, a workman, who, after seven years' occupation in this branch, at the age of 22 1/2 no longer owned a single tooth. According to KUNERT, the conditions are bad amongst confectioners. On an average, they have at forty years of age, instead of twenty-two,

[1] FEILER, 1913 p. 15.

seven sound teeth only, *i. e.*, 78 per cent. of their teeth are lost or carious».

In this citation of FEILER (some of whose conclusions probably need correction), chlorine and sugar are expressively given as factors of caries. Sugar we knew before, but chlorine was perhaps not so known. The strongest chlorine-former which we »civilized» people consume is, however, common salt. We know that NaCl quickly dissociates into free chlorine and Na. The dissociation-constant of NaCl is, according to BOIS-REYMOND[1], 84 per cent., *i. e.*, of 100 molecules eighty-four dissociate into free ions and functionate like free molecules. That sodium chloride should be an objective factor of caries is surely astonishing, but, theoretically, it is obvious. This assertion is practically confirmed by what is written above concerning the chlorine and also from our knowledge of the malady scurvy, which is to be regarded as a pure salt-disease. NaCl is, in fact, a poison, to which the human body accustoms itself in response to a successive increase of the dose. It is said that the Japanese, who, like all Orientals use very little common salt, sometimes take this salt when intending to take their lives.

Sodium chloride is osmotically one of the strongest preparations which we consume. The effective strength depends upon the concentration. We know, however, that many people consume enormous quantities which do not at all stand in proportion to what the human body really needs.

»MICHAELS states that the most active dental caries is found in the mouths of hypo-acid individuals, *in whom saline chlorid predominate* over the acid elements of metabolic waste etc.» [2].

This stuff is used not as seasoning only by the North-Arian people, but also as a preservative. All fat and

[1] BOIS-REYMOND, 1913 p. 215. 100 mol. NaCl = 16 mol. NaCl + 84 atoms Na + 84 atoms Cl.

[2] BURCHARD and INGLIS, 1912 p. 339.

much meat are consumed in the form of salt-preserved food. Saltpetre, which is an osmotically strong salt, is also a preservative. In the last few decades the consumption of (preserved) meat and fat has been very much increased amongst the North-Aryan races, which together with the increased consumption of sugar explains the simultaneous spread of caries.

That the human body needs some of the inorganic salts is clear, but in a very little quantity, and possibly as organic compounds only. The consumption of common salt amongst the North-Aryan people and the orientals differs enormously. It is even said that non-Aryan people living in cold latitudes need very little salt, and there are European colonists in Siberia who never use salt in dressing their food. Animal food itself contains a certain percentage of common salt.

The human body is able to accustom itself successively to all salts and poisons by gradually increasing the ration. And the more the body gets the more it likes to have. What is the cause, however, of the consumption of so much common salt by the North-Aryan people? Evidently first the climate, which asks for strong animal food, albumen and fat, which is very often — as regards poor people, as good as always — preserved, *i. e.*, salted. But just albuminous food needs in its turn »strong» gastric juice, *i. e.*, much HCl, which we get by eating common salt. Certainly the inherited desire for albuminous food is a factor not to be forgotten. »Civilized» people in our latitudes as a rule regard a dinner without meat as no food. To go to the opposite extreme and become a vegetarian is in the author's opinion also an excess, but to diminish to a minimum the quantity of preserved meat and fat given to children is certainly wise.

Between sugar and salt we can find the following

Parallelisms.

1. Salt is osmotically about ten times stronger than cane sugar and five times than grape sugar (in the same concentration).

2. Sugar is eaten by many people in enormous quantities, especially at a young age (time of eruption and after). Moreover sugar is commonly kept in the mouth for longer intervals than salt.

3. At this age, salt is relatively less consumed.

4. Salt dissociates and in this way nearly doubles its osmotic strength.

5. Sugar does not dissociate (cf. HÖBER [1]).

6. Sugar is harmful only when passing the mouth.

7. Salt is separated by the body and transported away by the glands standing in the service of external secretion, *viz.*, the saliva-glands. In that way a part of the salt (Cl) consumed passes the mouth twice.

8. Dentine *in vitro* is permeable to NaCl (Cl), only slightly so to sugar; dentine *in vivo* seems to be absolutely impermeable to sugar and *probably* to salt also. The gums are under favourable conditions permeable to NaCl (according to OVERTON [2] impermeable, and possible penetration of the gum inter-cellular). Red corpuscles are impermeable to sugar, permeable to Cl (anion of NaCl).

9. The organism seems, in the authors opinion, to have a certain affinity for NaCl, which can easily lead to over-consumption. (This opinion is rejected by OVERTON [2]).

10. By diffusion (cellular or inter-cellular) NaCl is able to trophically annihilate the gingiva, generally developing an infiltrated wound shown by experiments *in vivo* and empirically known in scurvy and in that way favouring the arising of root-caries.

11. The osmotic strength of NaCl increases towards

---

[1] HÖBER, 1911 p. 240.

[2] Personal communication.

the end; the contrary is true of grape-sugar (LAZARUS-BARLOW [1]).

12. Against a membrane impermeable to NaCl, the osmotic pressure of the blood is about twice as great as against a membrane permeable to NaCl (because about half of the salts of human blood is NaCl).

13. About 1 per cent. salt solution and 10 per cent. cane sugar solution are both the average minimum for hypertonic effect upon the blood (cf. Chapter V). The relationship between the possibility of the formation and that of the formation and presence of a 10 per cent. sugar solution probably depends upon the age of the individual; the latter (sugar) is favourable in the young, the former (salt) in a more advanced age.

14. Salt-colloids (animal albumen and fat) seem to be rather tougher, more insoluble and stick faster to the teeth than sugar-colloids (sweets). (The opinion of the author, perhaps not correct.)

15. People who daily eat strongly salted food, but little(?) sugar, *viz.*, the fisher-population in Scandinavia, have very affected teeth.

16. The enzyme of the saliva, the diastase, is active only in presence of chlorides, especially NaCl (MICHAELIS [2]).

17. The osmotic strengthening of the saliva by addition of sugar is raised after 2 minutes, of salt after 4 minutes (s. Chapter V).

18. The presence of common salt (like many other neutral salts) in so weak a proportion as 0.4 standard solution (= 2.34 per cent.) increases the reversible power of hydrochloric acid by 26 per cent., which means that the formation of organic acids and grape sugar is correspondingly increased, if HCl and NaCl are present.

17. Presence of sugar in the saliva stops fermentation processes (s. among others MILLER [3]).

---

[1] LAZARUS-BARLOW, The Journal of Physiology 19. 1895. 153.
[2] MICHAELIS, 1913 p. 75.
[3] MILLER, 1889 p. 39.

Medical and Other Preparations.

Besides salt, sugar and saltpetre, we must include all the chemical condiments, which now begin to flood the markets more or less of all civilized states. They may be divided into such preparations as are expressively stated or given as chemical substitutes for seasonings, drinks, jams, etc. and those which are pure adulterations, *viz.*, sacharine. That these preparations do not play so subordinate a rôle is fairly certain, but to what extent is not within the knowledge of the author.

Among objective causes of caries, we must also include medical preparations which are soluble in saliva. From the point of view of their power to do harm to the teeth, we may mention those that are given for long periods, such as salicylic, iron, bromic and mercuric salts, etc., which produce caries, when given shortly after eruption. At a later age they stimulate the carious processes already at hand. This applies to crown-caries; to the beginning of root caries these substances are always effective. Generally, all these salts partly pass the mouth twice, being inorganic salts. Empirically we know that salicylates as well as iron-preparations destroy the teeth, the different effect in mouths being ascribed to the duration of the treatment as well as to the structure and hygiene of the mouth, just as in the case of other objective factors. How mercury cures devastate mouth and teeth every specialist in venereal diseases knows. Haloid salts are theoretically in general the strongest, but in practice the concentration, of course, is decisive.

In order to fix the connection between »caries and drugs» Auston [1] has experimented with various salts and compounds of mercury, lead, bismuth, silver and copper. »Although it was found that the drug was partly excreted into the oral mucose, yet, it is rather an open question whether this excretion at one time may be so long continued

[1] Auston, Brit. Med. Journal 1910. 621, ref. Marshall, 1917.

as to accelerate or even cause any deleterious effect upon the erupted teeth» (MARSHALL [1]). As is seen from the citation, AUSTON and MARSHALL only think of the possibility of drugs being able to damage the teeth *direct.*

Mineral waters are certainly no blessing to humanity. Children in any case must be quite saved from these drinks prepared by carbonic-acid. The consumption of carbonic-acidulated waters (including ale) has thrust aside natural water, which is able to dilute the secretions of the glands, in that way counteracting to a certain degree the necessary consumption of harmful materials.

The natural mineral waters of the European continent are all practically *hypertonic* (caries-generating) to blood, in undiluted concentration, and have just, on this account, a special effect upon the stomach.

Acids and Alcohol.

The organic acids have all a low osmotic pressure, but in great quantities they are, of course, able to increase the total strength of the saliva. Their principal influence is connected with their property of dissociating H-ions and in that way stimulating the chemical and physical reactions.

Sugar and salts are certainly not able to enter living dental tissue and the carious dentine. If they were, diffusion of salts would begin and the osmosis cease, *i. e.*, the plasm would not be osmotic influenced further. There are, however, other preparations which theoretically have a great osmotic strength, but according to our experience do not exercise any influence upon the carious process. We mean the alcohols, of which, for our part, ethyl alcohol only can be considered. What can be the cause of the inability of alcohol to influence caries? According to physical chemistry we know that ethyl alcohol is an agent which has a very strong power of entering animal cells and tissues. Thus, if alcohol has been proved to be quite

[1] MARSHALL, The American Journal of Physiology 1917. 225.

negative as regards caries, it must mean that alcohol (which, of course, on account of its easy solubility is quickly transported from the mouth) is able to penetrate the carious matter and diffuse into the pulp, thus not exercising any osmotic power. Our knowledge of the property of alcohol as a remedy in our practice seems to confirm this supposition. It is probable that some of the organic acids, *viz.*, fruit acids, are also able to penetrate and operate like alcohol. This pretention that the arising of caries is independent of alcohol is contradicted, however, in the statistics given by v. Bunge [1] concerning caries in abstinent and non-abstinent Mohammedans, which give as result that the former under quite the same(?) conditions have double the number of carious teeth.

But, probably, this statistical fact is only apparent and is ascribable to the circumstance that the alcoholic liquids in discussion contain a great deal of *sugar;* or it might perhaps be explained in this way, that the non-abstainers consume food which more resembles the occidental. In any case: statistical »facts» must be very carefully controlled by check-researches. General experience does not correspond to the observations of v. Bunge.

Summary.

1. On logical grounds we claim that *natural* food and drinks cannot cause a disease, far less a destruction of the most resistant animal tissue, the teeth.

2. We know empirically that sugar and chemical substances are agents in the development of caries.

3. To the food eaten by »civilized» people, especially by the North-Aryans and their transmarine descendants, large quantities of sugar and chemical materials are added. Such substances are also directly consumed in the form of condiments or medicines.

---

[2] v. Bunge, Schveiz. Vierteljahrschrift f. Zahnheilk. 1905.

4. As shown in this and previous chapters an objective factor alone cannot cause caries.

5. When dissolved in saliva chemicals and sugar develop an osmotic strength which under certain circumstances is able to affect the tooth.

6. Dissolved materials which are able to develop an osmotic superpressure may under certain conditions force the plasm of the tooth up against the dento-enamel junction.

Criticism.

The first part of this chapter indicates the *analytical* way in which the author arrived at a definite opinion as to the nature of the objective factors.

These factors, although *not directly belonging to* the idea of *»food» — refer to substances generally mixed in the food* and so the notion of different forms of food as the cause and origin of caries was created.

This circumstance explains why the opinion of the objective causes capriciously varies with different writers.

In different works we thus read the following, logically contradictory, claims.

1. The food is responsible for the origin of caries.
2. The food is not responsible for the origin of caries.
3. Carbo-hydrates (sugar!) are responsible for the origin of caries.
4. Albuminates and fat (NaCl!) are responsible for the origin of caries.
5. Acids from the food cause caries.
6. Alkalies from the food cause caries.
7. Not mastication-favouring food is the decisive cause.
8. The constituents of the food are of subordinate importance.

However, we are able to reconcile these apparently contradictory opinions with one another by giving our attention to the difference between the notion: *»unspiced* food» and the general idea of »food».

CHAPTER VIII.

Subjective or Constitutional Factors of Secondary Caries.

In previous chapters we have made clear to our readers what the idea of »Secondary Caries» may involve: a chemico-parasitic process, following an *already formed* path direct to the centre of the tooth. The ancient theory of the genesis of caries was that »a stagnation of the liquid of the tooth» takes place; the modern opinion is a chemico-bacterial invasion. As a matter of fact, it is just both these processes in reciprocal action which together form the state of caries: a carious loss of dental substance. The former state is the primary; the invasion the secondary. This is the rule; the exceptions — »artificial caries» — are not numerous.

We are now going to consider the subjective factors of secondary caries, *i. e.*, the different kinds of entrances which are necessary conditions for a chemico-bacterial invasion.

These entrances may be: —

1. Traumatic.

Such an entrance may only result in secondary caries if the injury is so deep or large that the pulp is killed, exposed or seriously disturbed. This is often confirmed empirically in our practice. Even very deep traumatic fractures, involving a loss of up to one-third or even a half of the crown of the tooth, do not give rise

to caries, although decay may be present elsewhere in the mouth. The author has in his collection a tooth which by some injury lost one-third of its length about eight years ago, but which crown was without caries when cut off, *although the broken furrow was very deep and irregular and although the tooth was afterwards attacked by root-caries.* The explanation of this case — it may be mentioned en passant — is absolutely inconceivable by any other theory than that here advocated.

2. Operative.

If we make an entrance in a tooth for the purpose of filling or prosthesis, and the filling, inlay or pivot falls away, or the cement is dissolved, thus leaving a *deep* entrance open, the slow, chemical annihilation process will generally take place. The result is, however, not the same in different cases.

a) *Caries previously present.* In the case of a cavity prepared on account of *caries* the original caries canal, or parts of it, terminates in the bottom of the cavity. Along the remainder of this canal the destruction proceeds, if the situation is favourable, *i. e.,* if the filling has been lost or typically cervical defects exist. New primary caries may be formed, if the pulp is living, and the filling is quickly undermined by the subsequent softening of the dentine. In the case of a dead pulp the continuation of the process depends upon, how large a part of the former canal still remains and is accessible to the saliva, *i. e.,* where the defect of the cavity is situated. If in the upper part of the crown, the possibility of the process continuating (»recurrent caries») is very little; if, in the cervical part, it depends upon possibly remaining parts of the original caries canal. In other words, cervical defects of a filling are far more exposed to secondary caries than crown defects.

b) *Caries not present.* We extirpate, for instance,

the pulp of a sound tooth and cement a pivot or inlay, which afterwards falls out on account of the dissolving of the cement or from other causes. A chemical annihilation process will then begin, which successively softens the dentine. On investigating the cavity in this case, we shall find that the carious dentine *quite lacks its specific colour* and is most often soft, gray and easy to remove. (Exception, of course, in the case of smokers). Thus we have here a chemical annihilation without connection with »primary caries», made possible on account of a deep operative injury.

3. Prosthetic.

At the exposed root-necks of dead teeth it may happen in very favourable cases — weak, incomplete structure of the teeth and a denture lying close to, or comprising, the neck or the crown of the tooth — that a superficial erosion (a chemical necrosis) will be formed, successively developing into a cavity. Caries is in such cases, however, very often a question of latent caries becoming active. Such latent neck-caries is sometimes difficult to observe on account of its faint colour. If the tooth is extracted and dried it appears clearly and splitting will, of course, immediately show the anastomosis with the pulp. In the case of an erosion caused by different dentures, under the protection of which the intensity of the fermentation and catalysis reaches a maximum, which would be inconceivable in other circumstances, we have the most typical kind of *artificial caries,* under which head, *in the case of dead teeth,* »traumatic» »operative» and »prosthetic» caries jointly come. Fenchel has proved that »in all probability» the dental substance can be decomposed by electrolytical dissociation (= formation of mineral acids?), produced under protection of dentures and clasps of metal (ref. Äyräpää. [1]). Cut-

[1] Äyräpää, Sv. Tandl. Tidskr. 5. 1912.

off roots lying under dentures show the typical state of artificial caries.

4. White Decay.

So-called »white decay» is superficial acid-erosion, which may go on, or stop, *depending upon whether or not primary caries has been formed under it.* In the former case the erosion obtains the characteristic colour showing that primary caries is present. »White decay» belongs in the main to very young teeth — the facial cervical sides — and to wisdom-teeth. Its origin is probably to be ascribed to very weak and incomplete structure of enamel which has never reached its consolidation state, together with an excessively bad state of the mouth; also to very early or very slowly erupted teeth. In the latter case it lies in a pocket of overhanging gum where the conditions of fermentation are very favourable. »White decay» is, however, most often a superficial softening(?) or structural incompletion of the enamel only, forming a more or less porous layer under which the enamel is quite hard and is as often *not* »affected» by caries. We might claim — in referring to the latter case — that (fresh) saliva exercises a certain hardening (neutralizing?) influence upon the enamel, (cf. MILLER [2]).

»White decay», finally, surrounding a central carious area is a sign that the enamel in this zone is undermined by the caries-canal, *i. e.* the plasm is stagnated in the dento-enamel junction.

5. Primary Caries.

In 999 cases out of a thousand the formation of secondary caries or pathological loss of dental substance is identical with *microorganic* liquefaction *of a primary caries*

MILLER, 1889 pp. 157 and 171. cf. with
HEAD, Dental Cosmos 1907. 802.

canal. In favourable cases — early contraction of the pulp, good state of mouth and freedom from osmotically strong saliva — this canal may have a great power of resistance against destruction, forming chronic caries, but, hypothetically, it will be first softened by the acids (HCl; HNO^3) and then penetrated by microorganisms, which latter probably live upon the plasm lying in the tubules.

As we have shown before, the caries canals are of two kinds; crown caries canals and root caries canals. In adults teeth quite free from one of these states are very rare. Therefore, if natural teeth are put in a mouth for artificial purposes they will become carious: their secret canals succumbing to chemical and microorganical destruction. As long as the pulp lives, the closed, latent canals are generally safe, but a killing of the tooth often means that they will be opened, *i. e.*, secondary caries beginning. After the death of the pulp root caries canals change into open cavities more readily than crown caries canals. The author has seen natural teeth which have acted as artificial crowns for twenty years without caries, while the mouth in question during this time has shown plenty of new cases of caries. The matter depends upon whether the root part of the tooth is carefully removed or not. Thus caries in a natural tooth used artificially is simply the recrudescence of a latent canal already at hand, perhaps unobserved, or in any case regarded as a harmless *superficial* colouration, when, in fact. it is a way through the dentine to the pulp chamber. That Miller and others have found caries in such teeth and microscopically identified this process with that in direct caries is thus not at all remarkable. Cf. however:

»Under the microscope the tubuli between these striations (= the borders of the »Zone of transparency» or caries canal) are filled with irregular grains or long pieces.

In dead dentine I have not been able to discover any analogous substances..... I was the owner of 300

human teeth, which have been used as artificial teeth. Nearly all of them were carious in different degrees. I cut 60 of them, but found in only one case an appearance like »Transparency», and in this case the possibility was not excluded that the transparency had already appeared during the life of the tooth.» (MILLER [1])

Summary.

In this chapter we have made clear the difference between natural, non-natural and artificial caries.

Natural caries:	Primary and secondary caries in *living* teeth after an entrance of histolological nature or exclusively caused by acids.
Non-natural caries:	Primary and secondary caries in *living* teeth, the entrance being of a traumatic, operative or prosthetic kind.
Artificial caries;	Secondary caries in *dead* teeth, the entrance being of a traumatic, operative or prosthetic nature.

Criticism.

It seems evident that the systematization of the different forms of a disease is an essential condition for fixing its etiology. We cannot say, however, that odontology has made any effort in this direction. Yet, in this complicated destructive process such a systematic seems to be more necessary than is usually the case. As repeatedly insisted upon, man complicates the problem to a great extent by operating in the mouth.

A general view of the systematicis given later on.

---

[1] MILLER, 1883 pp. 122 and 123.

CHAPTER IX.

Objective or Salivary and Microbic Factors of Secondary Caries.

I.

Oral Acidity in General.

To a thinking odontological investigator it must seem improbable, as many times maintained, that the »system of nature», which we regard as the fundamental cosmic law, should be quite lacking in the human mouth. For, we cannot reasonably accept the postulate that milk, fruits and bread, the very means of nourishment which nature has given us as our principal food, should be transformed into materials which are able to annihilate the most resistant organic tissues. It must *a priori* be excluded that diastatic and reversible fermentation of the (unspiced) food normally belong to the mouth to such an extent and power as to be harmful to the teeth.

The organic acids which appear in the human mouth in any large quantity are chiefly fruit, lactic, acetic and fatty acids.

Intact teeth, sections of teeth, carious teeth etc. were submitted to numerous tests with these acids, alone or mixed, in different concentrations, 1 + 1000, 3 + 1000, 4 + 1000, 1 + 10000, etc., and although the acid was frequently renewed and the tests lasted for months and years, there was no other effect than a very weak superficial erosion. This, despite the fact, that these tests *in vitro* are very favourable to possibly etching acids, as the

neutralizing effect of saliva is absent. It should be remarked that weak alkalies gave the same result.

A tooth split in half was kept in concentrated lactic acid from Oct. 2nd 1914 to June 6th 1916. The test-tube was then filled with a *white* porridge. The tooth was taken up, washed and dried. Only a superficial corrosion was to be seen under the corroded layer. The structure beneath was macroscopically unchanged. MILLER [1] says that his test ended in a superficial erosion which under the influence of different foods and substances, *e. g.*, coffee, tobacco, tea, etc., acquired different colours like those of caries. But such an »artificial caries» has nothing to do with the *real carious cavity*, which is characterized by a little regularly formed entrance leading to a deep cavern filled with a tough red-brown substance having peculiar properties and containing iron and blood.

We cite, however, BRUGNATELLI [2]: «Allo stato attuale delle nostre cognizioni, dobbiamo dunque ritenere con MILLER che non vi é alcuna differenza fra la carie prodotta artificialmente e la carie naturale, *salvo la mancanza di quella zona di transparenza della dentina, alla quale accennai parlando della vitalita.*»

It is to be observed that when speaking of different special acids, we must think of these in reality as a mixture, where the single acid is one factor only. Thus it is more correct to use the word »acidity» than to mention a range of different acids.

Objective, not living, factors of secondary caries are those which are able to chemically dissolve the carious matter and the caries canal. This fact does not need further comments. It is enough to refer to what is written above. It will be remembered, how great a resistance carious matter is able to exhibit against acids, stains, and solvents of organic substances. Of course, when speaking of the

---

[1] cf. MILLER, 1889 p. 153.

[2] BRUGNATELLI, 1913 p. 53.

carious matter we do not mean the disorganized part, but the tough, clear light-red matter which forms the upper layer of the caries canal, as well as this canal itself.

Is carious matter an organic or an inorganic tissue? According to the text-books it contains about 25 per cent. inorganic matter and consequently 75 per cent. organic. The strongest solvents of organic matter that we know are: xylol, toluol, benzol, chloroform, ether and turpentine. In spite of carious matter being regarded mainly as an organic tissue and, further, as a product of destruction, it is — contrary to expectation — *not soluble* in these substances, in any case not perceptibly so after many months.

We must, therefore, turn to the inorganic solvents, hoping there to get a better result. Beginning with the organic acids, which are the crux of the Millerites, we shall, however, find that diluted organic acids have no effect at all upon carious matter. Even *concentrated* lactic acid needs many months to dissolve a little piece of carious matter. (cf. KELLER's opinion. [1])

Consequently we must have recourse to the inorganic acids. It has now been shown that diluted HCl and HNO_3 slowly melt the carious matter. If a carious tooth be boiled in aqua regia, the interesting observation will be made *that the last remaining part is the caries canal.*

With regard to HCl and HNO_3, the former appears more or less in practice, the latter exceptionally in the mouth of a human being, and consequently the former absorbs most of our interest. If HNO_3 together with HCl were often to be found in mouths the formation of Aqua regia would cause a destruction and dissolving of, firstly, all ungenuine dental metal work. That in fairly frequent cases the formation of Aqua regia takes place (or perhaps, better: the ion-components due to its formation being at hand) is, however, without doubt.

---

[1] BURCHARD & INGLIS, 1912 p. 327.

Hydrochloric Acid.

That hydrochloric acid, absorbed by the saliva, is able to enter the mouth in different ways has been shown in a previous chapter. We should remember that these different ways are: (1) as dissociation product of sodium chloride from food and drink, (2) as product from rising gastric juice, (3) possibly in other chemico-physical ways, *viz*,, from acid salts.

HCl-Analysis of Saliva.

To detect the very little quantity of »free» HCl in the saliva is not easy [1]. The methods which stand at our disposal are;

Indicators: congo-red, tropäolin 00, phloroglucin-vanillin and benzopurpurin.

1. Congo-red paper is coloured blue by HCl. Organic acids, *viz.*, lactic acid and acid salts give no reaction.

2. Tropäolin dissolved in water gives a ruby-red to dark-red colour when free acid is present. It also gives positive result with lactic acid.

3. Phloroglucin-vanillin is volatilized together with the same quantity of the liquid under investigation, and then gives a light rose-red sediment.

4. Benzopurpurin in water solution gives with free HCl a light-violet colour.

Of all these reactions, of which phloroglucin-vanillin is the best one, we must say that they are not reliable for detecting *traces* of HCl, which is the important point here. If the test is positive, free HCl is certainly present, but a negative test is not a proof of the contrary. If the saliva contains albumen, peptone or salts in large quantities the reaction may be hidden.

[1] cf. Michaelis, 1914 p. 111.

The very little quantity of HCl contained in the saliva is not to be detected by qualitative analysis, and we must have recourse to quantitative analysis in order to obtain reliable results.

The methods used are:

1) Titration,
2) The »inversions» method.
3) Estercatalysis.
4) The gas chain method.

By titration we find not only the free (»actual») but also the »potential» ions (s. *inter alia* HÖBER.[1]) The last mentioned — the »gas chain method» — is probably too complicated for a common practitioner[2] and we must help ourselves with the other ones. The result of these is to a certain degree influenced by the presence of neutral salts, but for our purpose and in practice it ought to be as exact as we require.[3]

The »inversions» method can be adopted partly with sugar, which in the presence of a diluted acid breaks up into dextrose and levulose, *i. e.*, a catalysis takes place, and the acid is the catalyst. We can also use amyl acetate (CH_3COOCH_3) or diazo-acetic-ester ($N_2CH \cdot COOC_2H_3O$), when the method is called »estercatalysis».

The author has used the reaction with amyl acetate[4], which will be described here.

Theoretical principle:

The hydrochloric acid, if present, acts as a catalyst and forms from the amyl acetate still more acid, which latter can be measured. The reaction proceeds after the equation:

$$-\frac{dx}{dt} = k\ (A - x)^2 \quad (1)$$

---

[1] HÖBER, 1911 p. 141.
[2] MICHAELIS, 1914 pp. 117 and ff.
[3] cf. MICHAELIS, 1914 p. 85, cf., with HAMBURGER, II 1904 p. 499.
[4] Molecular weight 74.

where x = the quantity of acid formed.
t = the time required for this formation of acid.
$k(k_1k_2)$ = the constant of velocity (of the reaction).
A = the initial concentration of amyl acetate.

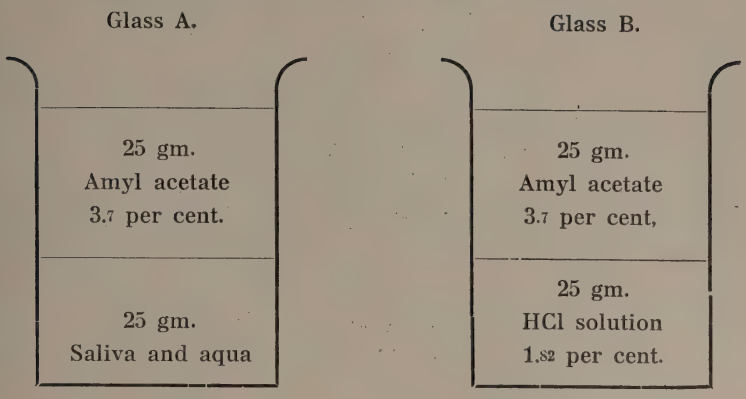
The derived figure (d) means that the equation applies to infinitely small quantities, *i. e.*, the chemical reaction proceeds without jumps. The minus figure means that it is a question of diminishing (the quantity of amyl acetate). The equation $-\frac{dx}{dt} = k(A-x)^2$ is by integration transformed into $k = \frac{1}{t} \log \frac{A}{A-x}$ (2)

Let a patient whose saliva gives a sharp acid reaction rinse his mouth carefully with 50 gm. aqua. Divide the quantity obtained into two equal parts. Fix by titration the quantity of acid present in the one part and note the figure (z). The titration can be executed by the help of phenolphtalein as indicator, this being the most convenient. On account of phenolphtalein giving evidence of carbonic acid, which *e. g.* methyl-orange[1] does not do, too high a figure may possibly be obtained, but as will be seen, any such fault may be regarded as eliminated.

The other part of 25 gm. (quantitatively, the »dissolved» quantity of saliva has practically »disappeared», because in the measuring a little is always lost on account of the liquid adhering to the sides of the measure) is placed into the glass beaker A. In the glass beaker B we put 25 gm. HCl solution 0.5 standard (= 1.82 per cent.), and in both A and B we then put 25 gm. amyl acetate solution, also 0.5 standard (= 3.7 per cent.).

[1] The converting of the colour of the methyl-orange is rather difficult to discern quickly in the opalized saliva solution.

Practical application:



We now let both liquids stand for 6—24 hours in a thermostat (50° C), the contents being stirred every now and then, but otherwise kept covered. For the investigation of the gastric juice four hours only are enough, but for the saliva, which contains very little HCl, a longer time is required.

After 12—24 hours we titrate the liquids in the beakers A and B and find respectively figures y and y_1 (quantity of acid). In glass A the quantity z (quantity of acid) was on starting fixed by titration, at the end we have now found acid = y. The quantity of acid formed during the test is thus $y - z = x_1$. In the same way we have in glass B $y_1 - 455 = x_2$.

From the formula we have the constant of velocity (k_1), when using saliva of *unknown* HCl concentration (C_x); and (k_2), the constant of velocity, when using HCl of *known* concentration (C_2), and get the following equation, the velocity of reaction being proportional to the concentration:

$$\frac{k_1}{k_2} = \frac{C_x}{C_2} \quad (3)$$

$$\therefore \frac{C_x}{C_2} = \frac{\frac{1}{t.} \log \frac{A}{A - x_1}}{\frac{1.}{t.} \log \frac{A}{A - x_2}} \quad (4)$$

but as the time (t) is the same for the two reactions, we get: —

$$\frac{C_x}{C_2} = \frac{\log \frac{A}{A - x_1}}{\log \frac{A}{A - x_2}} \quad \therefore \quad \frac{Cx}{455} = \frac{\log \frac{925}{925 - x_1}}{\log \frac{925}{925 - x_2}}$$

$$\therefore \quad C_x = 455 \frac{\log \frac{955}{925 - x_1}}{\log \frac{925}{925 - x_2}}$$

Ostwald [1] has drawn up a table, where the $\log \frac{A}{A-x}$ is swiftly found, when that of $\frac{x}{A}$ is known. We then get: —

$$\frac{C_x}{C_2} = \frac{\log \frac{x_1}{A}}{\log \frac{x_{,2}}{A}} \quad (5)$$

In the test in question;

C_x = the sought quantity of HCl (in milligm) in the saliva test (glass A).

C_2 = 455 mgm., the known initial quantity of HCl in glass B.

A_1 = 925 mgm., the known initial quantity of amyl acetate.

$x_1 = y - z$, where y = the titrated acid at end of test in glass A.

[1] Ostwald, Journal f. prakt. Chemie 29. 1884. 406.

z = the titrated acid at beginning of test in glass A.
$x_2 = y_1 - 455$, where y_1 — the titrated acid at end of test in glass B.
The quantity of HCl present in the mouth investigated is $= 2\ C_x$ (in mgm.).

Criticism: The result of an estercatalysis may be quite reliable from a practical point of view. Certainly, organic acids also produce catalysis, but in a proportion of 100—400 times less than HCl. The result is also influenced by presence of neutral salts.

The result of titrations and estercatalysis may be seen in the table, p. 176 and 177.

Conclusions.

The maximum proportion of HCl found by the author — up to 40 per cent. of the total acidity — was discovered in the mouth of an old man, who had all his teeth-crowns quite intact, while all roots were affected with deep gingival erosions. Of course, these two facts need not be related to one another, logically, but it is a probability bordering on certainty that the high acid-concentration and the corroded roots are reciprocally cause and effect in accordance with what is said in Chapter VI, the more so as otherwise the mouth was quite free from caries. The man said that he had to get up twice nightly and take potassium bicarbonate in order to be able to sleep. After half a year he again consulted the author, who then wanted to investigate his saliva, but the man stated he had now very little acid, adding: »I have followed your advice, decreased the consumption of salt and increased the consumption of water, and now I never have to get up in the night.»

It is however, experimentally fully proven that hydrochloric acid may appear in the mouth, and in quantities which are considerable. And as tests made before have shown that HCl is the mineral acid, *practically*

REGISTER COMPRISING QUANTITATIVE

Age.	State of mouth.	Time of day.	Quantity of acids (org. and inorg.) after titration.
69 ♂	All teeth present, firm, having no crown-caries, but all gingival erosions.	Early in the morning.	in mgm. 2·52
»	»	Before breakfast, but a little bread consumed.	2·19
»	»	After breakfast.	1·44
22 ♂	Four teeth missing; three carious teeth. Rather good conditions of mouth.	Before supper.	2·19
40 ♂	Six teeth missing. Very strong secondary caries (three molars broken down in nine months).	Early in the morning.	3·9
35 ♂	As good as all teeth having gold crowns, richmond crowns or fillings.	Before lunch.	2·19
35 ♂	Eight teeth missing, rather bad condition of mouth.	In the forenoon.	1·82
55 ♂	Very bad condition of mouth with loosening teeth.	In the afternoon.	1·99
c:a 40 ♂	All teeth present, all slightly loosening.	After breakfast.	1·82

General titrations gave as a maximum result 3·6 mgm. when figure; the same applies to methyl-orange, which shows that the

[1] Calculated in the following way: (Total titre quantity of acid ÷ result of general figure according to the effect of *organic* acids and made under the as- is further to be remarked that the calculation is based upon the atomic weight These would consequently need a fresh increase of the figure (2-4 times), but on indicator, which gives a result for carbonic acid also, which has a low atomic high, this fact will not influence the conclusions.

FIXATIONS OF THE ACIDITY OF SALIVA.

Quantity of free HCl after catalysis.	Calculated total acidity.[1]	Indicator.	Critical remarks.
in mgm.	in mgm.		
1·282	4·443	Phenolphtalein.	The acidity decreases during eating.
—	—	d:o.	
—	—	d:o.	
0·534	2·991	d:o.	
1·44	6·05	d:o.	Neurasthenic and bad conditions of stomach.
0·365	2·7375	d:o.	
0·865	3·117	d:o.	
0·328	2·682	d:o.	
0·341	2·33	d:o.	

using Phenolphtalein. Rosol acid as indicator gave a far lower quantity of carbonic acid in saliva is considerable.

Estercalysis) + 2·5 × Estercatalysis. This calculation is made in order to get a sumption that HCl is about 2·5 times chemically stronger than organic acids. It of HCl, which is generally about 2-4 times *lower* than that of the organic acids. account of the fact that for the Estercatalysis we have used Phenolphtalein as weight, we do right in ignoring this fact. If the figures obtained are a trifle too

appearing in the oral cavity, which is able to dissolve the carious dentine, we ought to be quite justified *in regarding the hydrochloric acid, possibly assisted by* HNO_3, *as the principal non-living objective factor of secondary caries.*

Concerning the concentration, this, of course, depends upon the quantity of saliva momentarily present. In the case of people having very little saliva, a rather small quantity of HCl will be comparatively more disastrous than a larger one with a rich saliva secretion. The result of the analysis is, however, the momentary quantity of free acid existing.

Other Acids Present in the Mouth.

That the presence of free HCl in the mouth, in consequence of its very harmful properties as *acid* as well as *catalyst* (10 to 400 times stronger than that of any organic acid) [1] and also on behalf of its property of being able to penetrate certain animal membranes, must play a more important rôle than the organic ones is evident. But if HCl increases the quantity of organic acids, these latter, on the other hand, dissociate and give off their H-ions, which with the chlorine from the dissociated common salt contribute to the formation of new hydrochloric acid, in that way presenting a reciprocal action with a gradual increase of the total quantity of acidity. The trivalent organic acids give off three H-ions, the divalent two H-ions of the same valency, etc.[2] Thus: if the saliva is positive or neutral, common salt may in some cases form HCl, in

---

[1] HAMBURGER, 1902 II p. 500: Coefficient of velocity of HCl = 1. that of lactic acid is 0.00901 and acetic acid 0.00345 (according to OSTWALD).

[2] On account of this property *tartaric acid* and *salicylic acid* are according to MICHAELIS (1914 p. 11) the most important of the organic acids in discussion. Lactic acid and acetic acid rank second.

some not, depending upon whether H-ions are present or not, *but in weakly acid saliva (= org. acids present) NaCl will always quickly cause strong acidity* (s. MICHAELIS. [1]).

As regards the presence of other mineral acids, nitric and sulphuric, we may mention that it is *possible,* as said, that also these acids may in rare cases and in very small quantities exist in the mouth. Nitric acid arisen by decomposition of ammonia [2] would then together with hydrochloric acid form the dangerous »aqua regia». Any greater *practical* importance they hardly have, for otherwise no precious metals would ever be lasting in the mouth and far less common ones. It is, however, interesting to read about a certain Mr. WATT in MILLER, who, like all other odontologists investigating this theme, was hunting for the mysterious cause of the colour of caries. He declared hydrochloric acid to be the cause of the white; nitric acid the cause of the yellow; and sulphuric acid the cause of the brown colour upon the teeth. As a matter of fact these acids do develop these colours upon dentine. [3]

That WATT'S suggestions cannot be correct, however, we can empirically prove. Experience teaches us that rapidly developing caries is lighter than slow. According to WATT yellow-brown caries is a product of the highest acidity.

MILLER has promulgated the thesis: »The microorganisms go before the effect of the acids.» [4] If this should perhaps be true of *organic* acids, it is, however, not at all applicable to hydrochloric acid. And in general, MILLERS *investigations apply to the organic acids only,* and he does not show any interest in this mineral acid, which, formed in another place of the human body, the stomach, rises to the mouth, where its presence is consequently known to him.

---

[1] s. MICHAELIS, 1914 chapter 50.
[2] s. MICHEL, 1912.
[3] ref. MILLER, 1889 p. 127, cf. MICHEL, 1912.
[4] MILLER, 1889 p. 139.

With reference to the question as to from where the *organic* acids come, we may state:

1. From acid food or drinks.
2. From reversible fermentation of the saliva enzyme.
3. From reversible fermentation caused by catalysts, *viz.*, hydrochloric acid.
4. From reversible fermentation caused by micro-organisms.
5. As secretion-product of the glands (the uric acid[1]).

The reversible power of salivary fermentation (diastase) is, as previously mentioned, in itself very low[2]. Only the presence of neutral salts and just chlorides, and among them especially NaCl, increases the effect. The fermentation caused by HCl has, on the contrary, no hinder and is without doubt one of the principal causes of organic acidity in the mouth. (The acidity formed by microorganisms is treated below.)

The acidity can be *facultatively* counteracted in three ways.

1. By dilution — abundant consumption of water.
2. By stimulating the glands producing alkaline liquids — the easiest way, by powerful chewing. Fresh saliva is certainly as a rule strong enough to neutralize all organic acids.
3. By consumption of artificial alkaline liquids — in certain sporadic cases necessary, but otherwise not to be recommended on account of other disadvantages accompaning these liquids.

Naturally the counteraction of acidity is derived from: —

1. The fresh saliva.
2. The decomposition of the oral liquid giving off alkaline products.

---

[1] Uric acid seems at a high temperature only soluble in saliva, and not always so.

[2] According to WOHLGEMUTH the diastase is quite ineffective in solution absolutely free from salt. s. MICHAELIS, 1914 chapt. 31.

It is finally to be observed that albumen-colloids have the property of storing (chemically binding with themselves) free H-atoms and Cl-atoms. Hence perhaps the natural explanation of the fact that butchers and provision merchants have very decayed teeth. They are commonly people who do not use artificial hygiene and the natural is missing on account of the excessive consumption of animal food. A permanent formation of the strong mineral acid may consequently take place in such mouths. This property of albumen-colloids does not apply to common salt, but this salt is *mechanically* bound up in the albumen (according to HÖBER [1]). »Fats deposited upon the teeth retard decay», say, however, BURCHARD and INGLIS [2].

Resumé.

1. The acidity of the mouth is principally a consequence of the presence of acid food and drink, HCl and NaCl.
2. The presence of HCl is chiefly due to NaCl and H-ions.
3. The presence of H-ions is mainly ascribable to organic acids and acid salts.
4. We cannot hinder organic acids entering the mouth, but the overconsumption of NaCl is easy to avoid.

Critique.

Even if the result obtained by ester-catalysis is not *absolutely* reliable — influenced *e. g.*, by neutral salts — the result is, regarded as *qualitative* analysis, a good guidance. HNO_3 and H_2SO_4 appear no doubt in the mouth in exceptional cases, but are in comparision with the first mineral-acid, HCl, for quantitative reasons of less importance.

---

[1] HÖBER, 1911 p. 164.

[2] BURCHARD and INGLIS, 1912 p. 328.

II.

The Microorganisms of the Mouth and their Relation to Caries.

The leading principles of the chemico-parasitic theory are:

»No caries without organic acids». »No organic acids without microbes.»

If we treat these principles critically according to the investigations and pretentions of MILLER we can raise the following points.

1. The presence of microorganisms of different kinds is a necessary and inevitable thing in human mouths, and PICKERILL's [1] comparative bacteriological researches of fifty carious mouths of town children compared with fifty caries-free mouths of »half-wild» Maori children yielded also the result that quite the same kinds of pathological and hypothetically non-pathological bacteria were to be found in all the 100 mouths. »There is no evidence yet adduced to prove that pathogenic organisms are active in producing caries. Though some of these are acid-producing, they exist in carious cavities only as causal visitors.» (GRIEVES [2]).

2. NONE of the specific dental microorganisms found have been cultivated and their specific dental harmfulness proved, *i. e.*, that they are a *cause* and not a consequence of caries. If the microbes found in carious dentine were the real originators of caries, *i. e.*, possessed the quality of being able to enter sound dentine, some of them could probably also be cultivated artificially upon sections of dentine. Let us see in what way MILLER's investigations confirm this fundamental question. He writes [3]:

»Upon sections of sound dentine the fungi *seem* in

---

[1] PICKERILL, The 6th Internat. Dent. Congress 1914.

[2] GRIEVES, Dental Cosmos 11. 1913. 1103. cf. also BLACK, 1908 I.

[3] MILLER, 1889 p. 64 and 132. Cf. also p. 50.

some cases to retain their lives for *a few days*, probably until the free organic substance is consumed».

Criticism a) The hazy words »seem», »some cases» and »for a few days» really give no reliable information in proof of the fundamental principles of this theory, and without doubt these words moderate or hide the truth *that these tests quite failed, although as they concerned the fundamental point in* MILLER's *theory, they certainly were both lasting and thorough.*

b) Why should not the microorganisms be able — *ceteris paribus* — to *enter* the tubuli and consume the organic matter there at hand? (s. BAUMGARTNER [1])

c) If the dental microbes could exist upon the organic matter of the dentine, *all the spaces of a tooth lacking enamel would acquire caries* (*viz.*, erosions, broken teeth, revealed necks etc.), if caries were to be found in the mouth.

d) One may be justified in regarding MILLER's attempts to cultivate dental microorganisms upon fresh dentine as having a *negative* result.

3) »Upon pure enamel no growth at all took place» (loc. cit., p. 64) and »Between the prisms of the normal enamel the fungi never enter», MILLER writes, (p. 132), which opinion BAUMGARTNER [1], however, rejects (p. 360). The same author regards parasites as being able to enter the enamel, if the structure is not *harmonically* built. If this supposition were correct no teeth could resist caries.

4. The acid-forming microorganisms die as soon as the substratum has received a certain acidity (loc. cit. p. 14). MILLER, however, assumes that the acid formed is washed away by the saliva, and that new acid is produced. The maximum figure for acidity formed *in vitro* by microorganisms, found by MILLER, was 0.75 per cent (found in what way?)

5. »In experiments on artificial production of caries

[1] BAUMGARTNER, D. M. f. Z. 5. 1911. 360.
s. FLEISCHMANN, Österr. Zeitschr. f. Stomatologie 5. 1913.

Miller found that the pabulum of the bacteria needed constant change, otherwise putrefaction resulted and decay ceased» (lecture at the Univ. of Pensylv. [1]).

The lactic-acid-forming microorganisms, the principal ones in question, produce, according to *modern* and quite reliable measurements an acidity up to a maximum of 1×10^{-5}. Miller's figure is thus undoubtedly wrong. A small addition of acids will be enough to momentarily stop the work of these microorganisms.

According to Baumgartner [2] an English author named Goadby regards fruit acids as a preservative against the development of acid-producing microbes, because the Sicilians have good teeth although they eat lemons in large quantities — in fact, a very reasonable idea.

Concerning the maximal-concentration of lactic acid, caused by microorganisms, the following citation from Michaelis is valuable.

»Da nun viele Bakterien saure Stoffwechselprodukte erzeugen, so lag die Frage nahe, wieweit eine Bakteriekultur sich spontan ansäuren kann. L. Michaelis und F. Marcora liessen Bact. coli in milchzucherhaltiger Nährbouillon wachsen und fanden, dass unabhängig vom Milchzuckergehalt und von der anfänglichen Alkalität der Lösung die Milchsäurebildung stets bis zu einer Wasserstoffionenconzentration $=$ (H:) $= 1 \times 10^{-5}$ [= acidity] fortschreitet. Dies ist aber gerade, wie wir sahen die höchste (H:), die das Bact. coli vertragen kann. Wir dürfen hierin wohl eine bemerkenswerte automatische Regulationsvorrichtung erblicken, die auch für die Gewebe der höheren Organismen zur Geltung kommen dürfte: das schädliche Stoffwechselprodukt, die Milchsäure, wird durch gewisse biologische Prozesse aus dem Milchzucker gebildet. Die Wirksamkeit dieser Milchsäure bildenden Agenzien hängt von der (H:) der Lösung ab, und ist derart, dass bei (H:) $= 1 \times 10^{-5}$ eine weitere Milchsäurebildung nicht

[1] Burchard and Inglis, 1912 p. 341.

[2] Baumgartner, D. M. f. Z. 5. 1911. 353.

stattfinden kann. Hierdurch schützen sich die Organismen vor der Bildung schädlicher Säuremengen.» [1]

5. The carbo-hydrates give off organic acids on fermentation, whilst animal food gives off alkaline liquids, which latter fact has given rise to a theory of alkalescency as the cause of dental decay. Even, however, if the former are perhaps quantitatively predominant, the latter develop a neutralizing effect, assisted further by the saliva. A mixed food — such as that of »civilized» people — will not generally as a final result be greatly affected by the indirect *chemical* action of microorganisms, whether in a positive or in a negative direction.

In cases of mixed food we have in a carious mouth the following *positive reactions.* 1) From the saliva; 2) from fermentation of albuminates; 3) from fermentation of the organic animal food of the microorganisms. *This latter case also applies to the gelatinous membrane (»gelatinoid matrix»), which, according to Black* [1], *protects the formation and action of acids upon the enamel.* Against these positive reactions (according to MILLER) there stands one *negative* reaction: Fermentation of carbo-hydrates.

---

[1] MICHAELIS, 1914 pp. 112—113. »Now, as many bacteria produce acid products of metabolism, the question naturally arises how far a culture of bacteria can acidify spontaneously. L. MICHAELIS and F. MARCORA cultivated bact. coli in beef-tea containing milk sugar and found that independently of the percentage of lactose and of the initial alkalinity of the solution the formation of lactic acid always proceeded up to a (H.) [= acidity] $= 1 \times 10^{-5}$. This is, however, as we saw, quite the highest (H.) that the Bact. Coli can stand. In this we may surely perceive a remarkable self-regulating contrivance, wich should also hold good in the case of the tissues of higher organisms. The injurious metabolic product, lactic acid, is produced from milk sugar by certain biological processes. The activity of these lactic ferments depends upon the (H.) of the solution and is of such a nature that when (H.) $= 1 \times 10^{-5}$ *further formation of lactic acid cannot take place* The organisms protect themselves in this way from the production of harmful quantities of acid.»

[2] BLACK, 1908 I p. 74. cf. BURCHARD and INGLIS, 1912 p. 329.

As regards the existence of these »plaques», we refer to the detailed criticism of BRUGNATELLI [1], terminating in the following words: »Allo stato attuale delle nostre conoscenze su questo argomento è assai difficile poter dare un giurdizio: mi limitero soltanto a dichiarare che la riproduzione artificiale della placche, ottenuta da WAUGH, non mi sembra una prova sufficiente a conferire a questa teoria un carattere scientifico» (p. 298).

6. The organic acids — the result of the microbic fermentation process — are able to do very little harm to the teeth. A superficial, white colouring, perhaps a superficial softening of the tissue, is all that *in vitro* with the help of comparativeiy *strong* solutions is attained. The author has made tests with sections and cut teeth having deep bored-in cavities and also small carious spots by placing them in different solutions (1 + 1000), (2 + 1000), (4 + 1000), (1 + 10000), etc., at blood temperature, but in no case has the slightest development of already existing caries or even new formation taken place.

Similar tests, repeated in renewed saliva acidulated in different concentrations and added to carbo-hydrates (sugar and the saliva taken from the same strongly carious mouth), have given the same result. Check tests with alkaline liquids (dil. ammonia etc.) gave an analogous result.

MAGITÔT (1867), together with MILLER [2], is the only odontologist who claims to have succeeded in artificially producing caries. The way of procedure is described as follows. Both let the acid work upon an isolated part of a tooth (section) and afterwards dried this part in the air, after which according to their claims the characteristic colour and form of caries appeared.

In criticising this test we may say 1) that this way of procedure is not nature's, as teeth in mouths are never dry, and the acids operate over the whole surface of the tooth. That, further, not a single other investigator has

[1] BRUGNATELLI, 1913 p. 261.

[2] MILLER, 1889 p. 154.

succeeded in artificially producing dental caries (= loss of dental substance, carious caverns). It is therefore astonishing that MILLER evidently relies upon Magitôt's result in spite of his own claims: 1) That acids alone, without microbes, cannot produce caries, 2) that microscopic researches (which MAGITÔT never undertook) only give proof of real caries existing, and 3) that his own attempts to artificially produce *real* caries failed. Under these circumstances we are justified in regarding the result claimed by MAGITÔT for his tests as rather hypocritical and misleading.

BUNTING [1] regards, however, also such superficial acid erosions as analogous with early caries (cf. also RYGGE [2]), a rather general opinion, but absolutely untenable.

7. As many times maintained, the source of the specific *colour* of caries has been a stumbling block for our many dental investigators. MILLER did not regard the chromogenic fungi as the direct cause of the specific colour of caries, but considered the latter more as the result of the work of the microorganisms combined. He mentions [3] that gelatine used as culture media for oral microorganisms gradually obtains a brown-black colour, but he makes the important addition that whatever bacterium used the result is the same. The property of dark-colouring on microorganic fermentation thus lies in the »culture gelatine» itself [4].

8. MILLER mentions that tobacco smoke completely sterilizes a subtratum infected by dental microorganisms (after smoking 1/4—1/3 of a light cigar). He then asks: »Why comes it then that smokers in general can get caries?» And he replies, »Because the smoke cannot enter the carious cavities» (l. c., p. 193). In the first place it is absolutely inconceivable that the *smoke absorbed by the*

---

[1] BUNTING, ref. Dental Record 10. 1918.

[2] RYGGE, Norsk Tandläge Tidskr. 1. 1918, and cf. *ibid*, parts 5; 6; 8.

[3] MILLER, 1889 p. 70.

[4] cf however GOADBY, Dental Cosmos 1900. 201.

saliva should not enter all nooks and corners of the mouth, and secondly, we may ask: How comes it that carious cavities under such circumstances can be *primarily* formed in the mouth of a great smoker? — as is really the case.

9. We know that when sound dentine is changed into carious it at the same time alters its composition in such a way that the former is left with (about) 75 per cent. organic matter and 25 per cent. inorganic. Thus, after the invasion of the dental microbes *living solely upon organic matter,* the 75 per cent. inorganic + 25 per cent. organic matter is transformed into 75 per cent. organic + 25 per cent. inorganic matter. There is but one solution to this riddle: *An addition of organic matter to the carious tissue must have taken place during (or before) the process.* We know from what is published in this work that this addition is due to plasm from the pulp filling the »carious» tubuli.

»Aus der Art und Weise der Lagerung der Bakterien im (cariösen) Gewebe kann mann schliessen, dass die Mikroorganismen im Gewebe selbst — mindestens zum Teile die Bedingungen für Ihre Existenz vorfinden» (Baumgartner [1]); cf. test by Miller p. 182 this work. »Aus dieser Ueberlegung geht hervor, das *dem* Eindringen der Microorganismen eine Veränderung in der org. Substanz unmittelbar hervorgeht (ibid p. 360).

Miller has made the following comparative calculations (l. c., p. 128):

187.2	cm^3	sound dentine	weigh	0.3600 gr.
187.2	»	carious »	»	0.0821 »
			Loss	0.2779 gr.

The sound dentine contains,

according to analysis, 72.1 %	Calcium salts	0.2595
The carious dentine 26.3 %	Calcium salts	0.0192
	Loss	0.2403

[1] Baumgartner, D. M. f. Z. 5. 1911. 370.

The sound dentine contains	27.9 %	organic matter	0.1004
» carious »	73.7 %	» »	0.0605
		Loss	0.0399

From these figures MILLER concludes »that the carious dentine has in all lost $^{7}/_{9}$ of its original quantity, the calcium salts $^{12}/_{13}$, the organic matter $^{2}/_{3}$; in other words, the carious dentine has nearly completely lost its supply of calcium salts, as only $^{1}/_{13}$ of the original quantity remains. From the organic matter the rather little loss of $^{2}/_{3}$ of the original quantity is to be noted, and without doubt this circumstance is chiefly to be ascribed to the direct effect of the microorganisms» (p. 129). This reasoning really stands in opposition to MILLER's opinion of microorganisms consuming the organic part of the dentine, which would first be consumed, or in any case at the same time. In fact, this calculation gives in all ways a wrong impression, being based upon the tacit understanding that 187.2 cm^3 carious dentine *had the same volume also as sound dentine* which conception is quite unfair and misleading, thus reasoning from volume to weight [1].

By using MILLER's starting-figures, but arranged and calculated in another way, we can, however, get (in approximate figures) the following tabula: —

	Total weight	Calcium salts	Organic matter
Sound dentine.....	80 mgm.	58.7 mgm.	21.3 mgm.
Carious »	80 »	19.5 »	60.5 »

Here we clearly see that in two equal *amounts of weight* of sound and carious dentine the destruction process terminates in an *addition of organic matters* up to three times, which

[1] It would be quite another matter if one were able to artificially produce caries; then one would take two equal volumes of sound dentine, transform one into caries and compare afterwards. But to speak like MILLER about *loss* of total weight of organic and inorganic substances *from this starting point* is quite incorrect, as the carious dentine has swollen.

figures are confirmed by the analysis given in our text-books.

10. Against MILLER's theory the objection has been urged that caries in some cases has not arisen in intact teeth the pulp of which show gangrenous decay. To all these and similar objections MILLER replies that no caries can develop because acids are lacking on account of the absence of carbo-hydrates. We note this and rejoin: we often find carious cavities with entrances which are hardly perceptible and impossible to pierce, but the cavity filled with carious matter is itself wide and deep; and we ask, from where do the carbo-hydrates come to the bottom of such a cavity, where destruction proceeds? Perhaps someone will reply: they are brought thither by the saliva. Impossible, we think; but if so we ask: is it easier for the saliva to transport dissolved carbo-hydrates *than absorbed tobacco-smoke?*

11. The development of the carious process is microscopically distinguished by a strong expansion of the tubules, accompanied by a simultaneous narrowing of the intertubular substance. In order to elucidate this MILLER[1] gives a peculiar explanation. He writes: »By the pressure in the tubules a small compression of the intertubular substance can without doubt take place, but to displace this entirely is not at all conceivable. As the fungi are not able to penetrate into this substance, *we must suppose* that the fungi form a *pepsinlike ferment, which is able to diffuse into and dissolve intertubular substance,* while the NEUMANN's sheaths are still intact.» As one sees, an easy manner »to explain what none can understand.» It may, however, be remarked that if the fungi could give off such a ferment, able to diffuse into and dissolve the inorganic part of the dentine, teeth with defects of enamel of any kind would in fact be standing quite defenceless against possibly existing caries, which, as we know, is not the case. The

[1] MILLER, 1889 p. 145.

existence of such a ferment is further rejected by other investigators, indeed, later on by MILLER himself[1].

12. Acids produced by microorganic fermentation of carbo-hydrates would be the real and sole cause of caries. Sugar also belongs to carbo-hydrates. Sugar has been regarded as a cause of caries since ancient times. How interesting is it then to read in MILLER'S text-book that an addition of sugar in saliva *stops the fermentation process of the fungi.* From our culinary department we also know that sugar is commonly used as a means of preservation against fungi. And we repeat, that the diastase is functional only in the presence of salts, especially NaCl.

Resumé.

1. No proof at all has been brought forward showing that the dental microorganisms are the *cause* of caries.
2. Their presence is a consequence of the destructive process.
3. Their existence in the dentine is further based upon a *supply* of organic matter.
4. This matter is *blood-plasm.*
5. If acids contribute to caries, *organic* acids produced by microorganic fermentation have a subordinate influence only.

Criticism.

The latter part of this chapter treats the question as to microorganisms being the cause of caries from a critical point of view only, which criticism is mainly directed against the deductions drawn by MILLER.

The *initial* breaking down of the caries-canal is due either to microorganisms alone or to these in conjunction

---

[2] MILLER, D. M. f. Z. 1907. 292. cf., however, PREISWERK, D. M. f. Z. 1902.

with acids — which of them is the more important factor, we do not know at this moment, but if acids contribute, only inorganic acids take an *active* part. — When cavities are formed the effect of the local acidose in the cavity is probably due to inorg. and org. acids cooperating, although the effect of the former is predominant.

If microorganisms alone are able to break down the caries canal forming the advance guard, the second query yet may be open — with reference to the fact repeatedly emphasized that the same tooth may have chronic as well as open caries — upon what secret, internal base is the resistance of the *latent* closed caries canals established.

CHAPTER X.

The Pathological Development of Caries.

Mainly from a Macroscopical Point of View.

The Formation of Primary Caries.

The pathological development of caries according to the theory set forth in this work is identical with the formation of primary caries and its transformation into secondary, *i. e.*, the successive degeneration of the physiologico-pathological caries canal into a devastated tissue.

If we consider a newly-erupted tooth in a young subject we know that its pulp is comparatively large and very full of blood, that the circulation in the tubules is active; we further know that these tubules often end deep in the enamel and that the enamel is not fully formed, but has small pits, fissures, defects and folds here and there. »The tubules will sometimes be found to terminate in the cuticula dentis» (v. BEUST [1]). The membrane between the end of a tubule and the external surface can thus in certain cases be very thin. We must assume that even such parts of the crown as lack enamel *at the time of eruption* are covered with cuticula dentis, for otherwise the circulation in the tubuli would not be limited outwardly. That thus Nasmyth's membrane covers the whole crown independently of the existence of erosions.

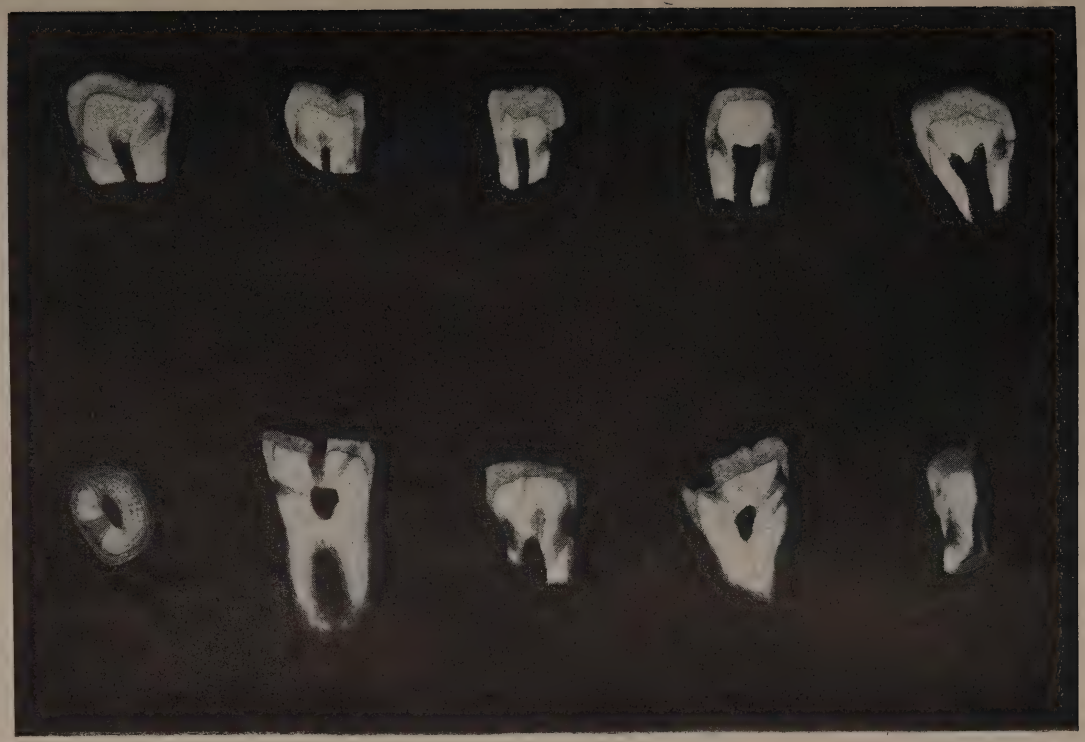
It is obvious that all these unfavourable circumstances are still more increased in an *early* erupted tooth in the case of weak, unresisting jaws. If we further claim that

[1] v. BEUST, Arch. f. Zahnheilkunde 1. 1912. 2. cf., also Chapt. IV.

through abnormal and unsuitable food, medicines or indulgences, the saliva has for a while acquired such a strong a concentration that it has a pressure greater than that of the pulp blood, a reciprocal action *must* then take place on account of known osmotic laws. As the living tissue does not directly allow salts and sugar to enter (according to general assumption, and in any case not sugar, cf. later remarks in this chapter) the result will not be a diffusion, but an osmosis, *i. e.*, the water of the pulp liquid (= the plasm) will slowly be pressed up to the region in action and the water of the saliva will *tend* to pass in the opposite direction. If the area is a small one, only a few tubules will be interested, but if it is, *e. g.*, an erosion, a rather large number of tubules are immediately occupied. For a short time only is the possibility of primary caries (crown caries) imminent. The tooth hastens to protect itself by contracting the pulp and simultaneously consolidating the exposed region by the formation of new dentine.

The plasm is, however — *ceteris paribus* — pressed up against the osmotic entrance of the enamel, which latter, as explained in another chapter, is in most cases of an histological nature. At certain repeated intervals the more concentrated saliva develops a physical action upon the liquid flowing in the tubules, from which it is separated by perhaps a very thin membrane, as enamel is lacking or structurally defective. The more concentrated liquid tends to equalise the concentration, according to the osmotic laws, by pressing or sucking to itself the liquid or the water of the other solution, *i. e.*, the liquid flowing in the tubules concerned. But this liquid will soon be occupied, the process of suction needs strengthening and the plasm of the pulp, which is standing in direct or indirect connection with the circulation in the tubules, is then drawn into the osmotic action. The tubules are, we may assert, successively filled with plasm, which becomes, if the sedimentation is left free from too strongly acidulated

Plate XIII.



Sections of Teeth with Caries Canals.

saliva, more and more resistant against all soluble matters (Chronic caries).

The pulp blood is in fact extremely sensitive to all kinds of irritation. We cite Squire [1]:

»The first sign of irritation is noticed in the blood supply. The blood-vessels are very numerous in the pulp, and render the tissue unusually susceptible to those pathological conditions which are associated with alterations in the circulation. First, by their arrangement; secondly by the structure of their walls; and lastly, by the character of intercellular substance through which they pass. The muscular tissue is almost absent and this formation offers very little resistance, if any, toward dilatation in even the largest arteries. This condition renders the tissue very liable to hyperemia and to inflammation.

The intercellular substance of the pulp is of a semi-fluid nature, and gives no support whatever to the walls of the blood vessels, and under such conditions it seems extremely difficult to avoid sensory irritation followed by vaso-motor stimulation in the reparative work upon the dentine.«

Tokle [2] relates a case in point. When excavating a central incisor of a 12-years' girl, he felt and was able to calculate the pulse in the tooth and compare it with the pulse at the wrist.

In a certain, greater or lesser, number of tubules the plasm is thus drawn up to the initial lesion, it may be a fissure, an erosion, a cingulum or any other histological defect, even an acid erosion on the exposed part of the root or on the crown, caused by a denture.

In this way it forms a horn-shaped canal in the dentine, the caries canal, which is easy to find macroscopically by a longitudinal section through the tooth, made in a such a way that the section is at a tangent with the

---

[1] Squire, Dental Record 7. 1915. 499.
[2] Tokle, Norsk Tandl. Forenings Tidende 1. 1918.

external caries-entrance and the pulp. It must, however, be carefully made if the canal is to be visible in its whole length (Plates XIII and XIV). Upon the surface round the carious area the enamel will sometimes look white and opaque. The reason of this is that the plasm, drawn up to the dento-enamel junction, often occupies an area far larger than the visible one. Consequently a great part of the enamel is undermined by the plasmoid sedimentation, which fact renders it non-transparent and whitish.

The Transparent Zone.

The general supposition is that the caries canal is a kind of »transparent zone» caused by external »irritation». In the first place it may be said that the property of transparency appears first in very thin sections, when the part concerned acquires a certain resemblance to new dentine like that found under the cusps. In thicker sections, on the contrary, the specific colouration may be clearly observed, which in the thinner cuts has nearly disappeared. Secondly, the property of transparency, as said before, is not at all exclusively associated with carious dentine, but is also characteristic of quite sound dentine. Otherwise the caries canal has genetically nothing to do with old transparent non-carious dentine, although our investigators have capriciously mixed them together. Now — as said — the general supposition is that this singular »zone» is a consequence of »irritation». Certainly, but what »irritation?» The explanation of this differs tremendously among different authors. Some (Tomes and Magitôt) regard calcification as cause of the transparency; others (Leber, Rottenstein and Schlenker) ascribe it to decalcification; Black regards the »hyaline area» as the earliest state of disorganization and Walkhoff, finally, has the conviction that an overproduction of intertubular substance has taken place at the expense of the odontoblasts

Plate XIV.

Longitudinal section of a premolar, showing caries canal. Drawn from nature.

Longitudinal section of a canine tooth, proximately showing two caries canals.

P.C.—Primary caries.

S.C.—Secondary caries beginning.

Drawn from nature.

and their off-shots. MILLER[1], from whom this is cited, accepts WALKHOFF's opinion and regards himself as having proved the untenability of the other conceptions, previously mentioned.

All these speculations have in reality very little value, because none of these authors had logically the correct starting-point, as nobody knew the fact that this transparent zone or caries canal represented a *constantly appearing and unconditional connection between the carious initial and the pulp.*

KANTOROWICS[2] speaks of »der Kegel» (the cone) as an expression for the caries canals.

We would further cite: — »Now, if the line of demarcation in the teeth affected by caries be the product of a deposit of lime-salts in the canaliculi; how, and by what means, are those salts carried to the line of demarcation? Is there any other vehicle but the blood to convey them?» (BECK[3]). This statement is very remarkable as the author expressively speaks about a hypothetical circulation of blood up to the carious zone, even if the suggestion of a depositing of calcium-salts here is not proven.

MILLER[4] writes: »I have found *iron*

1. On the surface of the enamel[5].
2. In the pulp.
3. In the enamel at the rim of the cavities.
4. Nearly constantly in the carious dentine. It forms a handsome blue line at the border between the decalcified and the normal tissue.

I am not able to give any explanation of this phenomenon[6].

---

[1] MILLER, 1889 p. 124.
[2] KANTOROWICS, D. M. f. Z. 8. 1910.
[3] BECK, Dental Cosmos 1898. 351.
[4] MILLER, l. c. p. 71. cf. with pp. 176, 177.
[5] Probably a precipitate from the reaction.
[6] Later on M. tried to give an explanation of the presence of the iron. (s. Chapter II).

MILLER has further investigated such a canal of »transparent» (carious) dentine and writes: »Under the microscope the tubules look as if *filled with irregular grains and pieces.*» Statements in the same direction, *viz.*, as to the presence of a peculiar substance in the tubuli of carious dentine are plentiful in MILLER's work. Page 150 (l. c.) he writes: »The appearance of rows of glittering irregular grains in the tubules is very often to be seen at the commencement of caries. Not infrequently sedimentation appears in the zone lying just beneath the carious zone.»

That these glittering grains can also be seen in natural teeth that have been used as artificial crowns, which fact MILLER states as proof that they cannot be a formation of the tooth itself, is, as shown before in this work (chapter II and p. 165), easy to explain.

MILLER's observations of these »pillar-like elements» found in the tubuli of carious dentine (s. Plate XV) are summarized as follows:

1. Not calcium, for treatment with H_2SO_4 did not produce the characteristical crystals of calcium sulphate;

2. Insoluble in organic acids. On the contrary, after treatment with lactic and acetic acids they appeared more clearly;

3. Insoluble in alcohol and chloroform;

4. Dissolvable in diluted H_2SO_4, »leaving behind a cornlike, hardly perceptible detritus» (p. 149).

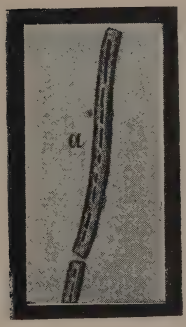
All these reactions (points 1, 2, 3 a. 4) are typical of old blood concretion.

5. »In diesen stäbchenförmigen Gebilden habe ich nie Mikroorganismen gefunden, dagegen habe ich dieselben wiederholt mit Kokken zusammen in stark erweiterten Canälchen gesehen.»

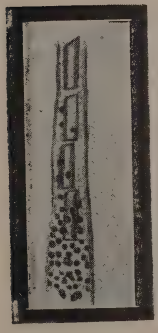
Concerning investigations of carious tubuli we would draw attention to the researches of KANTOROWICS [1]. He

[1] KANTOROWICS, D. M. f. Z. 8. 1910.

Plate XV.



Cylinder-like bodies in the tubuli of carious dentine.
1100: 1
(After Miller).



Cylinder-like elements in a tubule surrounded by microorganisms.
1100: 1
(After Miller).

treats of the existence of s. c. v. KORFF's fibrillæ, which according to K., appear well developed in carious dentine and are perhaps mistaken for the plasm threads in the tubules.

The Formation of Secondary Caries.

Let us return to the course of caries. We were discussing the formation of primary caries, *i. e.*, the physiological disturbance of the tooth, but not yet loss of dental substance. If now, however, the osmotic strength of the saliva continues to be active in consequence of receiving a further abundant supply of salts and sugar, plasm will continually rise up to the enamel, but on account of the fact that blood is coagulating a further supply of plasm will not pass through the upper layer, but will involve a larger and larger number of tubules in the action. The zone of plasm upon the surface of the tooth offers the microorganisms an excellent substratum and with the help *possibly* of mineral-acidulated saliva a cavity is formed (s. Plate XIV). Here, particles of the food are lodging, causing a process of fermentation, successively giving room to more extensive osmotic action and so on.

According to BAUMGARTNER's [1] and FLEISCHMANN's [2] investigations microbes should be able everywhere to enter »non-harmonically» built enamel — »la carie una Streptomicosi», BRUGNATELLI [3] says. We are justified in regarding this claim as an exaggerated one and due to the ignorance of the existence of the carious canal of plasm. But modifying this supposition as to the modus operandi, and claiming that microorganisms may conditionally or unconditionally be able to enter as fast as a canal of plasm has coagulated upon the surface of the

---

[1] BAUMGARTNER, D. M. f. Z. 5. 1911. 321.
[2] FLEISCHMANN, Österr. Zeitschr. f. Stomatologie 5. 1913.
[3] BRUGNATELLI, 1913 p. 340.

tooth (primary caries arisen), we must then ask ourselves how *chronic* caries in general can exist.

Either 1) the microorganisms would not have any desire for plasm from the tooth in question, which suggestion, however, is very improbable, as the same tooth can be seen having simultaneously chronic and open caries: —

2) This coagulum must in certain cases be able, with the help of the living element of the tooth, to undergo a strong consolidation in order to resist the micro-organisms; or finally: —

3) Not only microbes are necessary but also the assistance of highly acidulated (by mineral acids) saliva in order to be able to successively annihilate the caries canal.

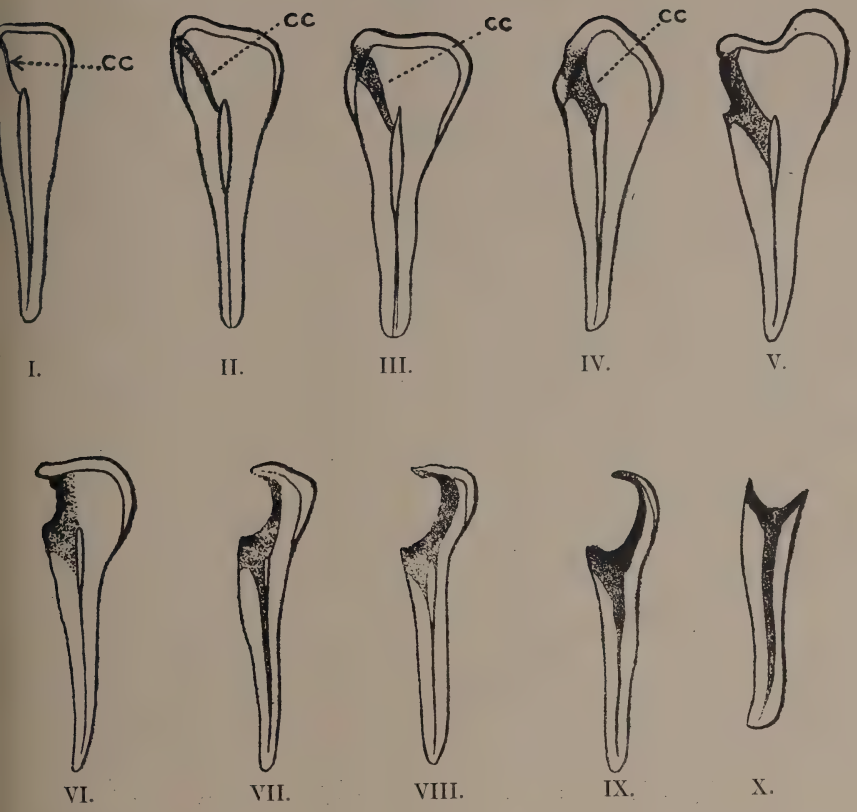
That a process of consolidation really takes place is apriorical, and otherwise confirmed by observations that chronic caries is transformed into open when the pulp is killed. Were this correct the oral acidose would only exceptionally play an important rôle, until the cavity had grown so large that food debris could in a larger quantity decompose and increase the acidity in the way treated in the previous chapters.

»In caries of the enamel we always find *with a striking regularity* as to the way and nature in which the microbes enter that: Those parts of the tissue are first penetrated (»durchwuchert») *which show the most abundant rate of organic substance*» (From BAUMGARTNER's [1] apparently very careful histological investigations).

The blood matter following the water, which flows outwardly, prevents on account of coagulation directly and indirectly the normal physiological function of the dentine (the nourishment and the formation of new dentine), and increases the percentage of *organic* material at the expence of *inorganic* which former serves as food for the microorganisms. This otherwise inexplicable situation now

[1] BAUMGARTNER, D. M. f. Z. 5. 1911, 361.

XVI.



Ten figures, drawn from nature, schematically illustrating the course of dental caries. The figures show the intimate connection between pulp and caries. With the annihilation of the crown the process has practically ceased; in any case the annihilation of the root takes a much longer time than that of the crown. In figure IX. the open process has reached the pulp, which involves its death. In I. to VIII. the pulp is fighting against the invasion. We see the caries-canal (C.C.) in its different states of development.

obtains its simple ground of explanation. The absorption of plasm relatively adds a rate of organic matter three times above the normal state. The laminae of dentine lying between the expanded tubules generally lose their support, swell on account of the absorption of plasm and fall into decay. The tubules become so wide that they nearly touch one another. »Neumann's sheaths» grow thicker. The uppermost layer of the carious matter is slowly liquefied by saliva, microorganisms and acids (HCl) and carried away, while more and more plasm is expelled — the larger the cavity grows, *i. e.*, the greater the number of tubules that terminate in the cavern. Slowly but surely the process nears the pulp, which tries to reduce the danger by building up a fresh wall of new dentine. Usually it will, provided no conservation takes place, be reached, invaded by microorganisms of different kinds, and broken down into a gangrenous state.

We have now given a short description of the course of *crown caries*, which differs from *root caries* in its time of formation, which lies within narrow limits (the period next to eruption, except for »artificial» caries only, *viz.*, caused by dentures, operative injuries, etc.), while root caries may begin immediately after the gingiva has receded exposing the root and then during the remaining life of the tooth; of course, subject to what has been said before about subjective factors of primary caries in general.

Mathematical Points of View.

If we study sections of carious teeth, carefully arranged in accordance with the extent of the development of the process, the sections *not being too thin* [1] and made

[1] If too thin, optical phenomena will affect the observations. Under great magnifying power the colour disappears, indeed, often without magnifying. The light is then also refracted during its passage through the dentine, which causes mistakes.

longitudinally in such a way that the caries canal is clearly visible, we shall macroscopically get a good idea of the course of caries.

Plate XVI shows schematically how the process is at first perceptible only as a band, a yellow tinge — in nature, of course, a tube — from the initial attack appearing as a brown area upon the enamel. This state is what we call primary caries and does not involve a loss of dental substance.

The process of destruction goes on and more and more tubules take part in it. But we notice that the intimate connection with the pulp is constant. It will also be seen how the process *at first* tends to conquer the pulp and, secondly, to destroy the tooth, or we may perhaps express it that the destruction of the crown is a consequence of the conquering of the pulp. We also see how the enamel is annihilated for the most part by undermining.

Let us ask ourselves how a chemico-parasitic process would go on taken from a proximate starting-point. The carious destruction is then *not* a reciprocal action between pulp and agents operating externally, but pure objective »decay», quite like the action of fungi upon jam, mould upon cheese, worms upon leaves, in exact conformity with the orthodox creed, with the exception only that the process is modified by the greater power of resistance of the enamel (s. Plate XVII). It is, however, to be observed that enamel, having a greater percentage of inorganic matter, would in reality be more quickly destroyed by *acids* than dentine. Plates XVII—XIX give a good idea of the course caries *would* have, if it were a pure objective process of destruction. As we see, the course is something quite different from what we know to be the case, irregularly and capriciously developing in all mathematical directions. It follows the layer of interglobular spaces according to the law of least resistance, and from here it

Plate XVII.

How proximate caries would develop if caries were caused by a chemico-parasitic process or by any agent operating only externally.

capriciously sends down here and there irregularly formed off-shoots, Plate XVIII.

How is it in reality? If we ignore more advanced states, when the destruction has nearly reached its maximum, the carious process shows regular mathematical and constant forms. Upon what factor does this regular development depend?

According to the osmotic theory the carious process is dependent upon three mathematical factors.

1. Initial point or external operation surface.
2. Direction of the tubules from this point or surface to the pulp.
3. The position and the dimensions of the *living* pulp.

We know then geometrically that the growth of the carious process on the sections is in the form of a triangle in such a way as to include more and more tubules in the action. In nature this triangle is, of course, a horn-like, conic figure (easy to prove by sections in different directions), with its top in the pulp chamber and its base upon the enamel.

Discussion.

If we picture to ourselves the process on a tooth which is deprived of its enamel and from which most of the soft dentine has been excavated, we should then see a figure like that illustrated in Plate XX a.

Have we caries in nature upon places quite free from enamel?

Yes, upon the root — so-called gingival erosion (Plate XX b).

Have these cavities always an identically mathematical appearance?

Yes, always.

Is this appearance like that of Plate XX a.

Quite, and a section through such an »erosion» will also show the same mathematical configuration and

connection with the pulp. If this connection is not so easy to see, allow the tooth to become quite dry and then the geometric configuration will sharply present itself.

Is this, the different appearance of crown caries, ascribable to the resistant enamel which hides the real configuration?

Yes.

But even if to a certain extent the enamel hides the clearly-defined mathematical configuration of caries, we know very well that proximate caries, especially on broad surfaces of teeth, shows in its early stages a round form, which afterwards takes on an oval one with the points directed lingually and buccally in order later on to go in apical and cuspidal directions. This elliptical figure tends to spread in the direction of the root, following the pulp. The labial cavity is also, when not too much advanced, of an oval form, but the points of the ellipse are directed mesially-distally. The explanation of both these facts is that proximate caries develops more easily in the direction of the root, following the pulp, but its development lingually-buccally *is limited to the width of the pulp*. It is easier, on the other hand, for the labial cavity to advance, at first, mesially-distally by occupying more and more tubules in a transverse direction. And the direction of the tubules diverges widely from pulp to labial side, which favours a development in a lateral direction, the more so as the development in a cuspidal direction is quite stopped on account of the process, standing in direct relationship to the pulp, not being able to go beyond a certain limit, *i. e.*, the region of tubuli starting from the roof of the pulp chamber. The process is limited by the direction of the tubules and the contraction of the pulp. It should be remembered that the *last remains* of »circular» caries (= far-spread labial caries in the front deciduous teeth) show the *cuspidal rim* of the tooth, of which the cause is now explained. In an apical direction the gingiva commonly raises a barrier.

Plate XVIII.

Longitudinal section of a canine tooth illustrating schematically how early caries would proceed if caused by an agent operating externally, namely, microorganisms, which would at first certainly follow the stratum of interglobular spaces, where the resistance would be least, and from there send out processes through the tubuli.

The growth and development of a cavity is thus controlled from the »active» pulp and not externally. The anatomic form of the entrance is, however, to a certain degree determinative. If it is a fissure, the course of caries must at first follow the same, and if a series of small laterally lying erosions becomes carious, *e. g.*, on a front tooth, the process will gradually terminate in a loss of the cusp of the tooth on account of undermining.

There is, however, no doubt that the carious process, when arising and lying on a plain surface, manifests a regularity and constant conformity, which would be inconceivable, if it were a pure objective annihilation process, but distinctly indicates the product of a reciprocal action between pulp and an externally operating agent, an assertion which grows to certainty on studying sections of carious teeth upon which we can see the constant relationship between caries and pulp (Plate XII).

The explanation of the mathematical properties of the course of caries lies consequently in the circumstances that caries follows the tubules the shortest way to the pulp and that the course and expansion is directed *from the pulp*. We have never seen a lingual or frontal cavity on a front-tooth or any other tooth going transversely through the tooth and forming a round hole right through it, which, if the process were a pure chemico-parasitic one starting simultaneously from a labial cavity and a cavity in *for. coecum* would be a common occurrence. The direction of the tubules is no hindrance, provided that the chemico-parasitic agents also tend to follow the tubuli, because after the contraction of the pulp the tubuli from the lingual and buccal sides meet one another in the former place of the pulp.

The Pathological Constitution of the Caries Canal.

What does the carious canal signify? We know:

1. That caries is an annihilation process.

2. That from the carious point of origin to the pulp a weakly coloured red or yellow zone is always to be seen.

3. That this coloured area of the tooth is *much more* resistant to acids and stains than the fresh dentine. (A fact proved by the author and confirmed by CAUSH[1].)

4. That the invading harmful process, by whatever it may be caused, has an object, negatively seen — that of dissolving and annihilating the dentine, — and will of course *never* harden or render the tissue more resistant *than it was before.*

5. That consequently this hardened and coloured area of the dentine, forming a zone down to the pulp, *must be a product of the tooth itself.*

6. That this hardened part of the tooth always terminates in the pulp wall, has the colouration of plasm, contains iron and blood matter and follows the way of communication between the carious point and the pulp — the dentine tubules. It must, consequently, in the name of human reason, be a product of the pulp, *i. e.*, represent a flow of pulp plasm through the dentine-tubules up to the point of caries initial upon the surface of the tooth.

But what can be the driving force of this stream of plasm?

We know:

1. That in the tubules there flows a liquid which obviously must be of plasmoid character and which stands in connection with the pulp organ.[2]

2. That the blood in certain cases, *e. g.*, in that of trauma is pressed out into the tubuli, colouring the whole tooth red, which colour can also gradually disappear.

3. That the blood in general is very sensitive to physical influence.

4. That the pulp blood is also very sensitive to external influence.

---

[1] CAUSH, Dental Record 2. 1916. 57.

[2] cf. Chapter IV.

Plate XIX.

How labial caries would appear if caries were caused by a chemico-parasitic process or by any other agent operating externally.

How labial caries in reality appears.

5. That the walls of the pulp vessels are very thin and the other tissues of the pulp are very delicate.

6. Some histologists — Docent Fleischmann, of Vienna, and one of his assistants — have in a certain case found erythrocytes in the tubuli of a decalcified tooth hardened with formaline.[1] And it is evident that if red corpuscles are able to enter the narrow tubuli it must be much easier for plasm to go up, as the tubules are narrower than the erythrocytes (2—4 μ and 8 μ respectively).

7. That a tooth at eruption, especially, if this takes place too early, will have the pulp organ in active and close connection with the inside of the enamel.

Thus, the author's claim that plasm from the central vessels of the tooth can, under influence of physical processes, rise to the belt of the enamel, is thus free from objections.

The Properties of the Carious Dentine.

The reader's attention has already been directed to the qualities of carious matter, *i. e.*, the fresh, elastic, carious dentine in cases of living pulp, not the top, rotten, quite soft or decayed layer. These qualities are: —

1. It is as a rule odourless in teeth with a living pulp.

2. It is more resistant to acids than the fresh dental tissue (according to the author).

3. It is more resistant to stains than the fresh dental tissue (according to Caush[2] *inter alia*).

4. It contains iron (according to Miller, Cohn[3] and the author).

5. It contains blood matter (according to the author and his assistants).

---

1 Personal communication, Vienna 1913.

2. Caush, Dental Record 2. 1916. 57.

3 s. Chapter II.

6. It contains about 25 per cent. inorganic and 75 per cent. organic matter (according to the text books).

As a conclusion, it may thus be said that carious dentine is a tissue which has been transformed chemically and physically. This peculiar transformation is effected by the tooth itself and has teleologically seen a certain purpose. What purpose?

What is the *first* result of the pathological carious process according to this work? — The weakening and the killing of the pulp.

What is the *second* result? — The complete annihilation of the tooth.

But the tooth like many other organs has a great power of »self-preservation» *i. e.* reacts advantageously towards external stimuli. It is clear from what is written above that caries is a fight between some objective factor or factors, as the aggressive stimuli, and the pulp, as the reacting organ, and further that the carious dentine is a tissue transformed by the central organ of the tooth, the pulp, and in such a way that it now offers greater resistance to certain dissolvable matters.

Thus the carious dentine is transformed by the pulp, thereby attaining a means of protection.

Protection for what? In the first place, the body, and in the second, the tooth. In what way is the carious matter able to protect body and tooth? By the formation of a barrier: a barrier, well to observe, not against the carious process itself, as the tooth is most often condemned, but against a greater danger. It seems to be quite evident that the course of an annihilation process entering the human body cannot possibly become a means of protecting it. Certainly we know that microorganisms indirectly give rise to substances, which are toxic to themselves [1]. But these things are transported by and stand in intimate connection with the *blood*, and without

[1] *s. inter alia* SUNDBERG, 1915 chapter IX.

Plate XX.

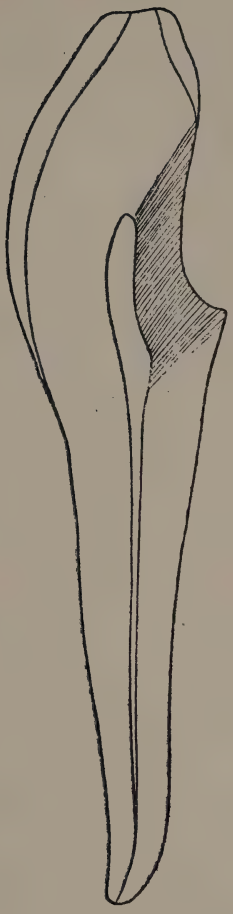


Fig. a.

Section of a tooth with caries, the rims of the enamel and the upper layer of the carious matter having been removed. Drawn from nature.

Fig. b.

Longitudinal section of an inferior front tooth, showing gingival erosion. The lines from the pulp to the defect were macroscopically visible. Drawn from nature.

the blood they would be of no use. Here we have just a channel of blood, and whether this blood contains any matters harmful to possibly existing microorganisms, even if very probable, we do not know, but the single fact that we have a stream of blood traversing a tissue, where no blood otherwise passes, is a fact sufficiently important to absorb our attention. That the tooth in certain cases, but not always, is able to stop the process of decay, we know, but what may be the protective design *when the decay is proceeding*. It should be remembered that the process has arisen through the influence of salts, sugar and other chemical materials, which physically affect the pulp liquid. It should further be remembered that this physical action is according to osmotic laws based upon the *permeability* or *impermeability* of the tissue, which forms a wall between both the active liquids, having, physically seen, the function of a »membrane». The relationship between this »membrane» and »caries» we are now going to develop.

Permeability and Impermeability of the Carious Dentine.

Also for the advocates of the general opinion that caries is a loss of dental substance, caused by an external agent, the problem as to in what degree foreign toxic materials are able to enter the centre of the tooth and from there the common path of the blood must be interesting. First we must remember that materials which have no or very little harmful effect upon the body when in the stomach or passing the alimentary canal, will have — injected into or in other ways entering the blood — a disastrous and poisonous influence upon the central organs. This applies to different matters, many neutral salts, spices, seasonings, CO_2, etc., etc. (see, *inter alia*, HAMBURGER[1]). According to the common idea of caries, the carious cavern is to be regarded as a direct entrance to the blood system

[1] HAMBURGER, II 1904.

and the body. In spite of this, if we ignore the researches of the bacterial invasion as such, none of our investigators — as far as the author knows — has fixed the slightest attention to this important point, and, yet, in their opinion matter of whatever kind should be able to enter. It may also be suggested that if the carious cavity was really such an open door, the pulp would be killed immediately after the carious process had affected the tooth. The pulp chamber of a carious tooth would then, as a rule, be filled with pus, and periostitis, cachexia and fever would always be associated with »caries». Almost nothing of this happens. If periostitis occurs it is, as a rule, at the end of the carious process, *i. e.*, when secondary caries has reached and destroyed the pulp.

Under usual conditions the process would take place in such a way that the water of saliva, possibly the salts also, would wander into the tooth, while the plasm would pass out. But the important fact must now be remembered *that the plasm is coagulating.* This condition changes the situation at first in such a way that a further pouring out or diosmosis of the plasm is hindered, and the plasm successively occupies a larger and larger part of the crown inside of the first layer of plasm; furthermore, it is very probable that the coagulum stops inorganic salts, in any case, sugar from entering or passing.

If, on the other hand, sound enamel, as Bunting and Rickert [1] claim, were permeable also *intra vitam* to salts of different kinds the simple consequence would be that immediately after eruption, when the tooth would, of course, be more accessible, an interchange (diffusion) would take place *everywhere through the whole crown* with a simultaneous wandering out of the pulp-plasm *i. e.*, a real loss of blood would take place from all teeth; in other

[1] Bunting and Rickert, The Journal of Nat. Dent. Ass. 5. 1918.

words, if this supposition were correct, man would simply lack teeth.

We can, however, claim, on account of empirical facts, that only in exceptional cases are toxic materials able to penetrate the cavern into the blood, passing the pulp chamber after the pulp is dead. With regard, further, to the peculiar properties of the carious dentine, which properties culminate in great power of resistance, yea, greater than that of the teeth in general, we do not hesitate to claim that it is just the carious tissue which forms such a wall. Not, we repeat it once more, against the carious invasion itself, but against the entrance of harmful matters through the tooth into the blood. Also the thickness of the undissolved carious matter is important, because the osmotic pressure is rendered weak by distance.

If carious matter were *permeable* to practically all substances (the carious matter being a destructive tissue according to the chemico-parasitic theory), it might mean the intrusion of impurities into the pulp with early death of the latter and the poisoning of the blood as a secondary effect, *but at the same time a swift equalisation of the osmotic pressure.*

If the carious matter is *impermeable* to harmful materials, it may mean that the body is protected against the invasion of toxic materials and against greater loss of plasm, but also subject to more durable pressure, *i. e.*, expulsion of blood, with (on account of the successive annihilation of the tooth) the plasm offering a good substratum to the microorganisms. On account of the exclusion of extraneous matter, an equalisation of the osmotic pressure will be *hindered.* In this we may see — if we desire — nature's preference of sacrificing the *teeth* rather than allowing a poisoning of the blood and the *body.*

The third possibility is, of course, also at hand, that materials in reality enter, but in very small quantities only, and that the pulp is able partly or wholly to

counteract the harmful effect. This eventuality implies that the carious dentine is only slightly permeable to the salts, and this will practically not change the matter in cases where the saliva obtains strong superpressure. The carious substance should consequently be a direct product of the osmotic process, which has formed a so called precipitationmembrane. Such a membrane is characterized by being permeable to *water;* impermeable to most other things, but especially to *those materials which have developed it.*[1] If this claim — that carious matter is a semi-permeable membrane — is correct, the carious matter must be permeable to water (*i. e.*, allow of osmosis or osmotic action), but impermeable to many other things and especially to the solid substances of the blood and, finally, to the materials which have formed the »membrane».

Although this reasoning is based on an assumption it is astonishing what a reasonable teleological proof it supplies — *ceteris paribus* — of this peculiar substance, the carious matter, and of the whole mechanism of caries.

Now, someone may object that if the carious matter is such an excellent wall against the enemy, how comes it that the process in general goes on? This is just the remarkable point. We asserted that the carious matter was a »membrane» which osmotic science has called semi-permeable and which is a product of different osmotic fluids when acting on both sides of a (at first) permeable membrane, the cuticula. But this semi-permeable membrane or precipitation membrane that has arisen by plasm settling in the dentine, is — as said — characterized by ts permeability to water, and consequently it forms no hindrance to osmosis. If such is taking place on account of the presence of osmotically active agents in the saliva, more and more plasm (blood water) will be pressed outward,

[1] See, among others, Bois-Reymond, 1913 p. 209. Blood-coagulum must be regarded as a semi-permeable membrane as being impermeable to *blood* (in spite of possibly opposite »official» opinions) and certainly to »saliva» also.

but neither the solid constituents of the blood nor harmful agents will penetrate the carious matter.

What are then the remarkable points in this physiological struggle?

1. The pulp works to render the distance from the surface of the tooth greater and greater.

2. If the pulp liquid is nevertheless pressed out on account of a porosity not yet being closed up, the pulp plasm forms with the dentine a membrane which is not able to completely stop the osmosis, but is, on account of its properties, able to hinder harmful agents entering the pulp and thereby direct into the human body and at the same to stop the blood corpuscles leaving the tooth. *In order to hinder a loss of blood and an invasion of toxic matters the animal body sacrifices the tooth,* but lessens at the same time the severity of the attack by the formation of the semi-permeable membrane, the carious dentine.

The struggle of the tooth for its existence is thus based upon the following facts:

1. The pulp is permanently standing in contraction — negatively seen. Positively, it is a building-up of new dentine at the expense of the volume of the pulp chamber.

2. The purpose is partly to consolidate the structure of the tooth, partly to increase the distance between the pulp and the oral cavity.

3. This latter fact is of great importance because the osmotic power, probably like all the powers of nature, stands in inverse ratio to the square of the distance, *i. e.*, a doubling of the distance with the same osmotic liquids means a diminishing to a quarter of the original power, or, which is the same thing, a quadrupling of the time; a three-folding 1/9, etc. In any case the osmosis will be slower in thicker membranes.

4, The plasm which is pressed out is absorbed by the dentine and gives it the great resistance of blood against physical and chemical matters (cf., the difficulty of removing old bloodstains).

The pathologically transformed dentine is preventing: a) Loss of plasm. b) Entrance of foreign toxic materials.

Positive Criticism of the Osmotic Theory.

1. The dentine has been experimentally proved to be sensitive and permeable to osmotic influence. Facts supplied by the author's investigations and later confirmed by CAUSH [1], BUNTING et RICKERT [2] and MANNING [3].

2. The enamel is certainly impermeable (cf., however, BUNTING (l. c.) and above in this chapter) but we know that newly erupted teeth have macroscopically visible, histological defects — consequently, of course, also microscopical ones — which later on grow together. The number and probably the depth of these defects increase the earlier the tooth erupts. Further, most of the teeth have permanent histological defects upon the enamel, *viz.*, fissures, cingula, grooves.

3. We know that a flow of liquid takes place through the dentine from the pulp, and it is further proven that the circulation-liquid of the body enters in and quite under the enamel (s. also Chapter IV).

4. The tubuli of the dentine must be:

a) either narrower when leaving the pulp and wider when reaching the enamel;

b) or, as wide from beginning to end, which latter is practically certain. In this case they start from a wall (the pulp wall) let us say, 1 cm_2 large and go to a wall (enamel wall) about 4 cm_2 large, radiating fan-like from the former to the latter. (It is not improbable that the

---

[1] CAUSH, Dental Record 5. 1915.

[2] BUNTING and RICKERT, The Journal of Nat. Dent. Ass. 5. 1918. Shown further at the Natural Scientific Congress of Vienna, Sept., 1914, Section Odontology. cf., also GOTTLIEB, Österr. Ung. Vierteljahrschrift 1913. 470.

[3] MANNING, Dental Cosmos 1. 1919. 21.

tubuli are slightly spiral-formed). In this latter case (b) the tubuli are more scattered towards the enamel. [1]

In both cases (a) and (b) a liquid flowing from the pulp to the enamel meets successively less resistance than in the opposite case, — a liquid coming from the outside and going to the pulp must cause a pressure and stagnation in the pulp chamber. This circumstance, which my experiments also confirm, as the coloured liquids osmotically penetrated the dentine much easier in the direction *from* the pulp than towards the same, fits in very well with the way of the tooth of saving itself, and with this theory. It is thus very easy for the pulp plasm to go up to the enamel, so much more so as a flow of liquid originating from the pulp plasm itself takes place in the tubules.

5. We know that between the pulp and every carious point or area of attack, if ever so slight, there passes a macroscopically visible, light yellow-red zone. This fact *must,* of course, have some positive significance. One may ask: does this weak, yellow-coloured zone constitute the path of a material which has entered the tooth, followed the tubuli and reached the pulp, as the result of an agent entering from the *outside;* or, on the contrary, of the pulp-liquid, the plasm, which has pressed its way up to the carious point of attack as the result of some kind of »irritation» lying outside; or, which is the same thing, has a reciprocal action between both taken place? [2]

It is evident that to a superficial observer the first opinion seems the simplest and most natural, and the general opinion also is that it [3] constitutes the result of a microbic invasion. But the fact that this zone *always* has the weak, yellow-red colour, the real colour of plasm

---

[1] According to MILLER (1889 p. 114) the tubuli are subjected to a senile narrowing.

[2] cf. FLAGG, Trans. N. Y. Odont. Soc. 1901, 108.
ref. BRUGNATELLI, 1913.

[3] = »zone of transparency», not, of course, regarded as a permanent »canal» between caries and pulp.

is alone sufficient to render this opinion untenable, for if it denoted the path of an invading product, it would, of course, *sometimes,* indeed as a rule, have a different colour on account of the different matters entering (as the caries canal is always standing in connection with an external *defect* upon the tooth), causing different chemical processes in combination with the saliva, the food, and the tissue of the tooth.

Even if we regard caries as a product of certain organisms having the special power of staining dental tissues with this specific colour, the path of the invasion must then be a way of decay through which other matters are easily able to follow, but in reality the carious part is, as CAUSH's [1] investigations also confirm, more resistant to stains than the sound dentine. In any case the colour would vary in different persons. *Actually there is always the same colour of plasm.*

6. In addition to the analytical and geometrical facts previously adduced in this work in proof of our claim, we will further submit a critical one. A front-tooth with Hutchinson's defects and having besides proximal caries was split. The proximate caries canals clearly appeared, but also some other »canals». It was soon clear that these latter stood in relationship to the Hutchinson's defects, where no (open) »caries» appeared. Consequently the latter canals cannot possibly be the result of an external carious invasion, or microbic »irritation», according to MILLER's theory, as »caries» is quite absent, but must be formed by something which is pressed up to the erosion from the pulp by a special power. This »something» cannot on account of different reasons be anything else but blood plasm.

If these canals were not caused by a force acting from the saliva upon the teeth, but by a force solely existing in the blood or in the pulp cavity itself, *e. g.*, increased blood

---

[1] CAUSH, Dental Record 2. 1916. 57.

pressure, the consequence would be that the canals would be seen radiating out in all directions from the pulp. Further, as this hypothetical power in the pulp, or in its blood, must act independently of the external contours of the teeth, the light-red canals would go out capriciously and planless from the pulp, whereas in reality we find, firstly, that only a few of them are to be seen on each cut plane, and secondly, that they are in direct and distinct connection with anatomically and histologically weak parts of the enamel, the neck, the fissures, the fossa canina, the proximal sides, etc. This constitutes an incontestible proof that the canals are a product of a force, externally existing, but operating from the centre of the tooth.

7. What power may produce this effect? Let us collocate the following facts: —

Human *experience* (not the »theories») refers caries to sugar, medical preparations, »water» (drinking-water), mineral water, in rare cases to chlorine, mercury, etc., but never, as far as the author knows, to the food itself. Nearly all the substances mentioned above produce when dissolved a physical process called osmosis. This process implies the possession of a power by virtue of which an interchange takes place between two liquids separated by a permeable membrane. We also experimentally know that the tooth is sensitive to osmotic influence, *i. e.*, allows of osmosis under certain conditions.

8. It is conceivable that in certain solitary cases — trauma or something similar — the blood of the pulp might be pressed out by powers active in the body; and that such cases also occur when the erythrocytes have been pressed into the tubuli, we know, but such cases must be regarded as exceptions. A power effective from the pulp cannot possibly stand in relationship and correspondence to *external histological defects of the teeth, not even with different compositions of the saliva,* and if such a power strong enough to press out the blood of the pulp up to the enamel existed in the jaws, it would

also burst the gingiva and thus caries would always be connected with bleeding of the gum.

9. The theory that caries is an active process of the blood agrees with all ancient theories of the genesis of caries to be a »stagnation of the liquids of the tooth», as well as the inflammation theory, which latter, even if in many points not correct, yet contains as much of the truth as the modern orthodox theory.

The claim of osmosis as the secret driving factor in the genesis of caries consequently rests upon a logically and empirically stable base.

Negative Criticism of the Osmotic Theory.

If the assumption of the osmotic expulsion of the plasm forming a caries-canal in the crown of the tooth immediately after eruption, through which the microbic and chemical processes *afterwards* penetrate, were a pure invention, a product of fantastic imagination, how is it possible that the strictly logical consequences of this claim can lead to an abstract confirmation of empirically concrete facts, viz.: —

1. The explanation of the relationship between caries and the contraction of the pulp [1], which gives us the solution to a series of facts hitherto inexplicable, *e. g.*, the insensibility of the top of the crown to caries. Why caries always follows the pulp by developing and never penetrates through the tooth, *e. g.*, from the buccal to the lingual side.

2. The explanation of the regularity of the form of early caries.

3. The explanation of the gingival erosions.

4. The explanation of the appearance of symmetric caries.

[1] This relationship is as a matter of fact independent of any theory of the etiology, but stands in close connection with the laws of the osmotic theory.

5. The explanation of latent, inactive caries.

6. The explanation of the peculiar properties of the carious dentine.

On the contrary, a wrong logical starting point would swiftly terminate in crazy consequences, the fallacy of which would be evident even to a child.

For sake of comparison let us see what *logical* conclusions we can draw from the chemico-parasitic theory (MILLER'S, WALLACE'S, etc., and their modifications, purely chemical or purely parasitic ones):

1. The carious process — *ceteris paribus* — capriciously affects the teeth and groups of teeth, and symmetrical caries is inconceivable.

2. Because an entrance through the enamel is the only condition necessary for an invading to be possible, the process will — caries being present in the mouth — first and last penetrate *all* fissures, cingula, *erosions, broken teeth*, etc.

3. Chronic caries never exists, at any rate never in connection with active caries. A simultaneous appearance upon the same tooth is quite impossible.

4. The external contour (*i. e.*, the border of the cavity against the enamel) is always of a very irregular and capricious form.

5. The carious cavity not too young forms in an advanced state an open canal direct to the pulp and the blood system, and the carious matter is a transportable product of ready solubility, offering very little obstacle or none at all to entering substances.

6. If once invading the mouth all teeth, or nearly all, become successively carious.

7. The carious dentine — the last remains of the hypothetical microbic process — consists of a pathological tissue, where the organic matter is equal to, or in a minority to the inorganic, or quite lacking.

None of these consequences corresponds with the truth.

Summary.

1. The transformation of the dentine tubules into caries canals is brought about by an osmotic interaction between the tubular plasm and the osmotically active substances dissolved in saliva.

2. The destructive process of the caries canals is caused by an invasion of microorganisms probably assisted(?) by strong acidity from mineral acids.

3. Nature offers greater or lesser resistance against this microbic invasion.

4. As the pathological invasion is closely bound to the plasm-canal which in its order is controlled by the pulp, the consequences are that the growth and development of the bacterial invasion is determined by the position of the pulp and the direction of the tubules. This gives the anatomic explanation of the empirical fact that caries develops in the direction of the apex. For the same reasons the regular elliptical form of the carious entrance, when caries is not too far advanced, also gets its simple geometric explanation.

5. The carious dentine must be regarded as a transformed tissue, which transformation involves a protection of the body against the invasion of foreign materials.

6. *Caries dentium* (not artificial caries or gingival erosions) is to be regarded as a *plasmolysis* of the *tooth.*

Criticism.

The mathematical laws of the development of caries comprise *natural* caries only.

That acids contribute to the initial annihilation of the primary caries canal is an unproven supposition.

In this chapter the pathological course is mainly treated from a macroscopical point of view. These important

facts have been until now quite overlooked by other authors in favour of microscopical investigations.

Whether or not caries is to be regarded not only as a *plasmolysis,* but also as a *»hœmolysis»* or »erythrocytolysis» *i. e.* if caries involves not only a loss of plasm but also of red corpuscles, is so far an open question (cf., Chapter III)[1].

[1] The expressions, »hæmolysis» and »erythrocytolysis», are perhaps not *quite* adequate, cf., SUNDBERG, 1915 p. 181.

CHAPTER XI.

Structure, Pressure and Caries.

To point out the connection between hard mastication and good structure of the teeth (= early and swift contraction of the pulp) ought hardly to be necessary. There are, however, some facts concerning the mastication-pressure, which may be of interest.

By mastication-pressure we mean that pressure which the lower jaw exercises against the upper one, *not the resistance which single teeth can endure.* [1]

The pressure is transferred by the lower jaw which works as a one-armed lever. According to the mechanical law for levers, the pressure is greater the shorter the arm of the lever is. This is the theoretical proof that mastication pressure *increases towards the back of the mouth.* Nature's own confirmation is not difficult to find. In the dog and the bear the tooth which is intended to receive the greatest pressure is found innermost in the jaw. The same observation may be made in the case of the elephant, hylobates siamanga and apes in general, which all have the most powerful tooth innermost in the jaw.

It is, however, by no means necessary to go to the animal world in order to obtain practical and incontestible proofs of the truth of our pretention. The centre of pressure in the mouth of a five-year-old child lies at the place where the second bicuspid is to come, and here is also to be found the most powerful tooth of the set; the

[1] Eckermann, D. M. f. Z. 9. 1911.

second deciduous molar. But the child grows, the jaw lengthens. The central mastication pressure moves backwards and the sixth-year molar appears. For six years its large masticatory surface receives the maximum pressure until the lengthening of the jaw necessitates reinforcement farther back. The years after the appearance of the twelfth-year molar denote a turning-point, the jaw of many individuals ceasing to grow after this period, partly because of local reasons (caries), partly because of constitutional reasons. In this case the maximum pressure remains on the second molar, and no tooth at all, or else only a rudimentary wisdom-tooth, forces itself forth. In the normal case, on the other hand, the centre of pressure is removed to the normal position of the wisdom-tooth and a powerful third molar appears. Generally people place the food when chewing between the molars.

The Mechanical Principle of the Jaw.

The pressure is transferred by the lower jaw, which, apart from the question as to whether the jaw-joint is a pure or a modified ginglymus, works against the upper jaw — mathematically regarded as a one-armed lever directed by two systems of muscles — one upper and one lower. As the statical moments of one-armed levers are equal, the two systems of muscles must be of equal power, that is to say, the maximum resistance which the lower jaw has to overcome is as great in opening the mouth as in closing it. In other words, *in order to forcibly open a firmly-closed mouth as much force is required as is to be overcome in order to close the mouth under maximum resistance.*

The mathematical qualities of the lower jaw are illustrated in Plate 18 fig. a and b. The curve CWJ denotes the anatomic lever, the nearest mathematical expression of which is the line CJ. On this are measured out the desired points w (the wisdom tooth), m (the

second molar) and b (the contact-point of the bicuspids) to correspond with the points W. M and B. In order to obtain the relative powers of the levers CJ, Cm, Cw, they must all be transferred to a horizontal plane. The most suitable plane for this purpose is that fixed by the anthropologists and known as the »Frankfort-plane». This is imagined to be so placed as to cut the bottom of the orbit and the upper edge of the meatus. We obtain thus, as diagram (a) shows, five points of this plane, C_1, W_1, M_1, B_1 and J_1. By measurements it can be seen that the line C_1W_1 is exactly half the length of C_1J_1, and thus $Cw = {}^1/_2$ CJ. This can also be seen in diagram (b) ($CW = {}^1/_2$ CJ) although for perspective reasons not quite so clearly.

By anthropometrical observations — made by the author in about 200 cases — it will be found that this relationship — that Cw is with very small deviations half of CJ — is always *constant*, both in the case of alluvial and of diluvial skulls, in young as well as in old ones.

The lever from the centre (the jaw joint) to the wisdom tooth is thus half as long as the lever from the centre to the incisors (or the whole lower jaw) lever. But the power is in inverse ratio to the length of the levers, and consequently *the effective power at the wisdom tooth is double as great as that at the incisors.*

This relationship is, as already said, constant in all human skulls, and the following gnatho-dynamometrical law can be laid down: The masticatory pressure at the wisdom tooth, or, at this tooth's place in the mouth, is twice as great as at the incisors, *provided, of course, the teeth have their normal position and quality.* By a simple calculation the masticatory pressure at every tooth may now be fixed. The pressure at the second molar in the lower jaw is about 17/10ths of that at the incisors, while that at the first molar in the upper jaw is about 15/10ths. If, therefore, the pressure at the incisors is, say, 20, the other figures will be 34 and 30 respectively, if it is 23, they will be 38 and 34.5 respectively.

Plate XIX.

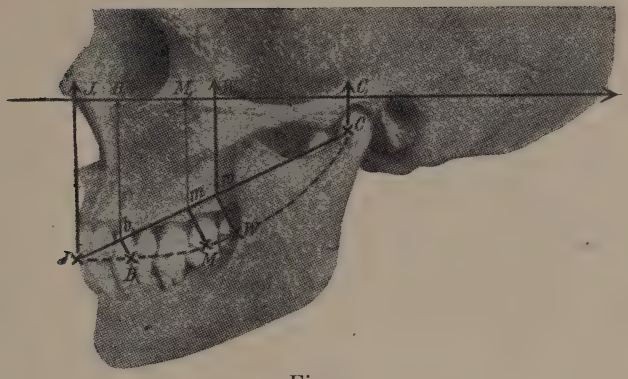


Fig. a.

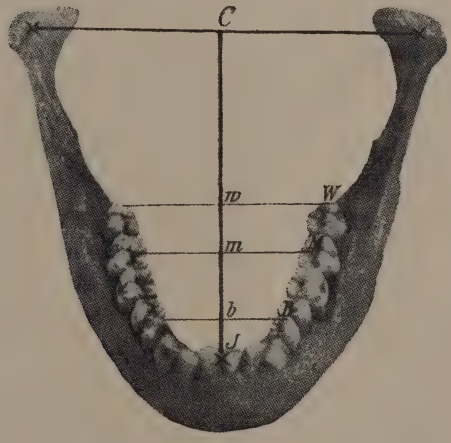


Fig. b.

The Gnatho-dynamometer.

The calculation of the masticatory pressure is an old desideratum. The significance of being able in every special case to calculate readily the maximum resistance is evident to every odontologist. Fixed starting-points and system in treatment would thereby be attained. Prosthesis could be based upon a solid scientific foundation, instead of, as hitherto, being founded upon chance observations. We have all had the experience of finding that some of our patients have bitten our dentures to pieces while in other cases the dentures, often quite contrary to our expectations, have shown great durability. There is a great difference between constructing a pivot-tooth for a pressure of 20 kilogrammes and one for 40 kilogrammes.

It would further be possible for the anthropologist to establish the exact masticatory pressure of different races and peoples, of town and country population, etc. It would also be possible to establish the declining(?) maxillary strength of the first, second and third generation of town dwellers.

When constructing a gnatho-dynamometer one must, of course, have a definite starting point. The nearest to hand is *the force exerted in order to press the teeth together.* It was in accordance with this principle that Black constructed his apparatus. This starting point is, however, erroneous inasmuch as at the forced biting together of the teeth one sinks the head and the pure masticatory muscles are strengthened by other systems of muscles, (particulary the occipital and cervical muscles and the sterno-cleido-mastoideus). The figure arrived at will consequently be too high. Circus artistes who lift weights with the teeth become most tired in their occipital and cervical muscles. Black [1] confesses, in fact, that he does not obtain the direct muscle-strength, but rather the strength of the peridental membrane. Black has further

[1] Black, 1908. I p. 170.

supplied his apparatus with springs, which serve as measures of force. But springs do not give any trustworthy result as measures of weight. In Sweden it is forbidden by law to use spring-balances in business. If a weight (dead weight) is put or dropped on a pair of kitchen scales supplied with springs, it will be found that the pointer goes up first, for instance, to 10 lbs., then down to 6, finally stopping at 8 lbs. If now we have a gnatho-dynamometer of spring construction, we *only* see the false maximum figure, inasmuch as the maximum power (live load) cannot be applied as dead weight, *but immediately after grows weaker.*

From literature one learns that attempts to measure masticatory pressure have been made by BLACK [1], SAUER, ROSENTHAL and THOURÉN [2]. Comparing the maximum figures which are given by the different authors, one finds that they are all below or about 100 kilogrammes, with the exception of BLACK'S, which reach to 180.

After many unsuccessful attempts to construct a gnatho-dynamometer the author proceeded from an opposite standpoint, namely that of calculating *the power required to open the tightly-pressed jaws.* From a mathematical and mechanical point of view this starting point is unassailable, but on the other hand it is obvious that in the case of so complicated a biological apparatus as a human jaw unforeseen circumstances create difficulties. It is conceivable, for instance, that the violent closing of the jaws may create an accelerated power, though this would, of course, be of minimum proportion, owing to lack of space. An attempt to measure such would probably meet with insurmountable difficulties. But for fixing the constant, or, if preferred, latent power of mastication, the above-mentioned starting point should be free from objections.

The appearance of the apparatus is made clear by

[1] BLACK, 1908 I.

[2] THOURÉN, Odont. Tidskrift 3. 1910. 298. cf., RIECHELMANN D. M. f. Z. 8. 1912.

Plate 20.

Fig. a.

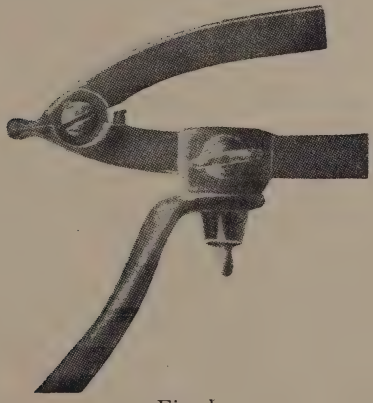


Fig. b.

illustration, Plate 20 Fig. a and b. It resembles a pair of tongs with two shanks moving about a pin. One of the shanks constitutes the long horizontal balance and is provided with a weight-indicator, the other, which is rotatory and detachable, is connected with a stand. The upper shank is, moreover, furnished with a movable loop-formed rider, which at its lower end can be loaded with three different weights. The inner part of this rider is provided with an edge which fits into the division lines scored out on the shank. These division lines are made after careful measurements and in such a way that the weight of the upper shank, with indicator and weight, will be balanced at the first division line, for instance, 10 kilogrammes.

The apparatus can be easily mounted on any operation chair. It may be fixed to the spittoon-bracket, being fastened there by means of a thumb-screw. The dynamometer can be turned in all three directions. The »bite-receiver», *i. e.*, the plates which are to be inserted into the mouth, have thin steel plates adapted to the front teeth. These are smelted over with soft metal. Just before being inserted they are further coated with a thick rubber tube, or nipple, which can be sterilized. Plate 20 b.

In order to limit the movements of the shanks when weights applied overcome the strength of the jaw, it is of importance to avoid a breaking force on the teeth. For this purpose a suitable tap or screw is fitted so that the shanks can only be separated 2 mm., which is sufficient to get a clear result on the indicator.

When measuring the masticatory pressure, one lets the patient sit comfortably in the chair and inserts the gnatho-dynamometer. Then weight No. 2 (standard weight) is placed on the cursor, but the balance must not be loaded until the patient has pressed his teeth together. When this has taken place and the patient is keeping his jaws closed with all his power, which is generally easy to control, the rider is placed on the balance and moved quickly over the graduated scale to the point where

one »feels» that the power is balanced. The plates thereby force the upper and lower jaw about 2 mm. apart. The measuring of the masticatory pressure is in this way very easy, and as correct as is practically possible. In case of superior protrusion the lower jaw should be pushed somewhat forward during the biting. Otherwise the result is a breaking power on the plates. To thoroughly remove this breaking moment is, of course, only possible if the biting is straight or the thickness of the plates is equal to zero. On the other hand it is to be noticed that just in consequence of a certain thickness of the biting plates the closed rows of teeth arrange themselves naturally for a straight bite. One cannot for this reason reckon upon an absolutely exact result for all jaws. For although the mathematical and mechanical position is not itself affected by the protrusion of the lower jaw, it is conceivable that a weakening of the muscles occurs owing to their extension. In cases of »superior protrusion» the masticatory pressure may therefore be measured at the bicuspids. An absolutely exact result is also impossible — irrespective of the mechanicals laws — on account of the different structures of the teeth and presence of diastema.

If it is suspected that the patient, commonly through fear of straining his teeth too much, has not exerted his greatest power, this may be verified by taking the pressure at, for instance, the first molar. There the patient usually exerts his full power quite willingly.

Once, the author measured the masticatory pressure of a lady and got as the result 20 kilogrammes for the front teeth. Suspecting, however, on account of the appearance of the powerful teeth, that the patient had not put forth all her power, the masticatory pressure at the first molar was taken and the figure 38 obtained. A calculation showed that the pressure arrived at for the front teeth must be 5 kilogrammes too little. Once again the pressure at the incisors was taken, after requesting the subject to use all her power. Sure enough, the pressure now went up to

Table.

Sex.	Age.	Pressure at Incisors	Pressure at Wisdom Tooth	Remarks.
Male.	48	24 kilos.	48 kilos.	First molars missing.
Female.	23	33 »	66 »	Complete set.
Male.	20	27 »	54 »	Complete set.
»	30	14 »	28 »	Molar region defective.
»	40	14 »	28 »	1 molar and 1 bicuspid missing.
»	42	36 »	72 »	2 + and + 2 never appeared, otherwise complete.
Female.	24	23 »	46 »	Complete set.
Male,	42	34.5 »	75 »	Molar region very defective.
Female.	41	22 »	44 »	2 molars missing.
»	26	10 »	20 »	Molar region defective.
»	26	24 »	48 »	Molar region defective.
Male.	27	5.5 »	11 »	Artificial incisors and bicuspids, evidently for little pressure.
Female.	55	22 »	44 »	Bridges in each molar region.
Male.	25	21 »	42 »	1 bicuspid missing.
»	31	29 »	58 »	All the lower molars missing.
»	25	23 »	46 »	2 molars destroyed.
»	24	22 »	44 »	Molar region defective.
»	56	39 »	78 »	Set nearly complete.
»	17	15 »	30 »	13 teeth missing.
Female.	24	19 »	38 »	Complete set.
»	35	17 »	34 »	Complete set (1 molar missing).
Male.	26	17 »	34 »	11 teeth missing.
Female.	27	17 »	34 »	2 bicuspids missing.
Male.	33	27 »	54 »	4 molars and 1 bicuspid missing.
Female.	16	15 »	30 »	Complete set.
Male.	34	24 »	48 »	Nearly complete set.
»	39	10 »	20 »	Bicuspid region defective.
»	32	33 »	66 »	All the bicuspids missing.
»	52	17 »	34 »	10 teeth missing.
»	30	33 »	66 »	6 bicuspids and 5 molars missing.
»	53	10 »	20 »	Extremely weak, loose, artificial teeth.
Female.	35	23 »	46 »	4 molars missing.
Male.	29	23 »	46 »	3 bicuspids and 5 molars missing, 2 bicuspids and 4 molars fitted with gold crowns.
»	23	29.5	59 »	2 molars missing.

By »defective» molar or bicuspid region is meant that the teeth are partly present but lack antagonists.

25 kilogrammes. A proof not only of the accuracy of the gnatho-dynamometer but also of the correctness of the gnatho-dynamometric law.

The author has drawn up a statistical table of 200 cases, of which thirty-four are submitted here [1]. From these one may draw the following conclusions.

Summary.

The masticatory pressure of women only in exceptional cases exceeds 30 and 60 kilogrammes, and of men 40 and 80 kilos respectively at the incisors and wisdom tooth.

The normal masticatory pressure of men is not under 25 kilogrammes at the incisors; of women not under 20. The maximum pressure the author has measured was that of a man of thirty-four years with 54 and 108 kilogrammes (119 and 238 lbs.).

The masticatory pressure and the structure of the teeth stand, without exception in direct relationship to each other and verify the law: the greater the resistance, the better the structure. *Between good structure and caries, on the contrary, no logical correspondence is exhibited,* which quite confirms the claims of Black [2] and Gassmann [3], who have investigated the problem from other points of view.

Criticism.

1. It is, of course, not impossible that the masticatory pressure could be individually increased, which would

[1] »Und zwar halte ich das Ergebnis der Messungen Eckermanns für viel wahrscheinlicher als die von Black und andern gefundenen Werte, wobei ich auch überzeugt bin, dass sich mit dem Eckermannschen Gnathodynamometer infolge seiner Einfachheit und des Fehlens von Federn einwandfreiere Resultate erzielen lassen.» Riechelmann, D. M. f. Z. 8. 1912.

[2] Black, 1908 I p. 170.

[3] Gassmann, Zeitschr. f. Physiol. Chemie 1908.

certainly have a great physiological importance. Whether this would also result in an increased power of resistance against caries, the future will show (cf., BLACK [1]). Systematic and comprehensive statistics might in this case teach us very much.

2. It is conceivable that the mouth-pieces of the gnatho-dynamometer could be lengthened and fitted with suitable »bite-plates» of, for instance, wood pulp, thus forming an apparatus for giving exercise to the jaws of children.

3. As regards the nature of the maxillary resistance itself, this is, of course, a product of time and the momentary pressure exerted, and may thus be divided up either into shorter time and greater pressure or into longer time and relatively weaker pressure, which in practice means that lengthy mastication with medium power entirely counterbalances less lengthy mastication with higher pressure.

4. The fact that good structure of the teeth is not able to hinder caries arising, does not, of course, involve that a development of strong mastication-pressure is superfluous from all points of view.

---

[1] BLACK. 1908 I p. 170.

CHAPTER XII.

Practical Conclusions and Prophylaxis.

I.

The theory of the osmotic origin of caries shows that caries stands in reciprocal relationship to the pulp. Conservative treatment has accordingly to accept the formulation of the following laws, which are based on a logical ground and are empirically confirmed:

1. In the young years of a tooth the pulp must be, if possible, preserved at any price.

2. In the middle age of a tooth the pulp can be removed without harm, *if all caries canals or primary caries are simultaneously closed.*

3. In the late years of a tooth the removal of the pulp is quite a tolerable procedure, and consideration need only be given *to possibly existing root caries.*

4. The cervical part of a (filled) cavity is the most susceptible.

The facts upon which these four laws are based are the following.

The work of the pulp is necessary to consolidate the tooth, as the tooth is not at all full-built on eruption. This consolidating means at the same time a contraction of the pulp in an apical direction and the more this process advances, the larger becomes the surface of the crown that is »immune». On the other hand, we see in this work that caries may be produced and lie latent, becoming active, if the pulp is killed or on account of

reduced resistance in general, produced by diseases, state of pregnancy, etc. these being the ways in which the tooth loses its power of resistance. If thus a tooth must be *root-filled* during the years in discussion, it must be carefully examined to ascertain if there is any latent caries in other places. In old age, on the contrary, the removal of the pulp is certainly justified, as regards the possibility of the loosening of the teeth. If we study mouths with loosened teeth (pyorrhoea) we shall find that any old root-filled teeth in the set will be standing quite firm. Exceptions occur only in such teeth as are over-burdened, this not being genuine(?) pyorrhoea. Root-filling only, however, is no effective remedy for pyorrhoea. The author regards the disease as caused by chronic inflammation arising through atrophy of the pulp due to osmosis.

As regards the kind of filling, the metal ones stand in the first rank as regard osmotic impermeability. In the second rank gutta-percha, not, of course, on account of strength, but on account of impermeability, and in the third place cement filling, phosphate or silicate, both of which are permeable to osmosis; a fact which may perhaps explain the frequent death of the pulp under such fillings. As regards amalgam fillings it is not improbable that these also are sensitive to osmosis when the surplus of mercury is not sufficiently pressed out. The reason for making this supposition the author obtained during an osmotic experiment, where the insulation of the tooth consisted of amalgam. Two days after (the setting) small balls of mercury were still pouring out (on account of the osmotic pressure?). A bottom inlay (*e. g.*, of gutta-percha) or lining under a silicate filling is always advisable. Food favouring mastication is from all points of view necessary. Hard and too frequent brushing, which must damage the soft tissues and lead to an exposure of the roots, must, on the contrary, be carefully avoided. Soft tooth-brushes, assisted by soap and tooth-powder, are to be recommended.

II.

It has many times been repeated that the concentration of the saliva for shorter or longer intervals during the eruption time and some years afterwards is the decisive cause of crown caries. In order to bring down this fact to the lowest possible practical point, the child must have very little salted food and not even be allowed large pieces of sweets (sugar) to suck (sugared food does less harm), and should have no long medical treatment (iron, salicylic, bromide, etc.), and, further, a rather abundant consumption of common water, which must be increased during medicine cures, is desirable. Finally, mineral-medical waters or other acrated waters with high percentages of salts and carbonic acid drinks are to be avoided. To people with fully built, caries-free teeth (no crown caries) all these things have very little or no effect at all until the state is reached when the receded gum tends to expose the roots of the teeth. *Then all teeth are again sensitive to osmotically strong saliva.* Provided the contraction of the pulp is so far advanced that osmosis cannot operate, the roots are immune, but, if this state is not reached, the salts and sugars are harmful to the same extent as during eruption. In cases of denuded roots a certain moderation must be observed in the consumption of salt, while recourse should be had to a rather abundant consumption of common water. We may say that, *practically,* (pure) sugar is the agent of caries to the young and common salt to the adult. That overconsumption of sodium chloride is at all ages a physiological poison to the human mouth is experimentally proved in this work.

The line between over and normal consumption may be quantitatively fixed in prophylactic order, cane sugar not exceeding 1—1.25 gm.; salt 0,05—0.1 gm. in solitary doses does not cause harm. [1] That is to say, one may theoretically

[1] Approximative figures, calculated in correspondence with the figures obtained in the cryoscopical tests as shown in Chapter V.

eat sugar (cane sugar) the whole day, 1 gm. (salt 0.05 gm.) at a time and one portion after the other, without harm, provided there is an interval of about 3 minutes between each act of consumption; — 5 minutes for sodium chloride.

To this comes a new factor, the temperature. As the osmotic pressure grows swiftly with rising temperature, all hot liquids containing harmful preparations are comparatively many times more dangerous than cold ones. A measured temperature of the food and drinks, especially for children, is therefore advisable. To a cup of tea or coffee holding about 100 ccm one must not add more than one piece of sugar (= 6 gm).

Summary.

Powerful mastication and abundant consumption of common water, a sparing consumption of salt, (pure) sugar, chemical preparations, aerated drinks and avoidance of too hot liquids and food are the leading principles of prophylaxis according to the osmotic theory.

Criticism.

Comparing, with respect to the harmful quantity of the objective factors, the chemico-parasitic theory with the osmotic one, we shall find as harmful in the former case an indefinite, yea, even a microscopic quantity (*»loci min. resistentiæ»*) of food belonging to our most frequent and natural diet; in the latter case a certain, rather high quantity of not absolutely necessary spices, while the consumption of a smaller quantity of these is not at all harmful. In the former case a meaningless state, in the latter one — *system*.

Systematic of Dental Caries.

Dental Caries=destruction of the teeth:

a) Physiologico-pathological state= **Primary Caries.**

b) Breaking down of substance by microorganisms = **Secondary Caries.**

I. *The carious entrance to a living tooth may be: —*

A) Constitutional or histological.

B) Exposed root on account of a retracted gingiva.

C) Acid corrosion or White decay.

D) Artificial 1. Traumatic
2. Operative
3. Prosthetic.

If any one of these entrances is present the result may be: —

Primary caries of different kinds:

a. *Crown caries* or

b. *Root caries,*

both of which states terminate in:

1. *Chronic caries* or develop into
2. *Secondary caries.*

If the carious entrance is of the kind mentioned in points A), B) and C) we speak of: **Natural Caries.**

If of that kind mentioned in point D) above we speak of: **Non-natural Caries.**

II. *The carious entrance to a dead tooth may be:*

A) Traumatic

B) Operative

C) Prosthetic

If there exists any one of these entrances (and the sanitation is bad) the consequence will be a slow, purely chemical destruction: **Artificial Caries.**

III. Entrance to the exposed *root* of a *living* tooth under certain conditions: —

A) Living tooth

B) Exposed root

C) Very contracted pulp (down to the root)

D) High acidity of the saliva

E) Osmotic exchange between pulp and saliva:

Gingival erosions

Criticism.

The fact that operations are performed in the mouth renders the problem of caries more complicated. Whereas in other parts of the body we are concerned with physiological or pathological states, we must in the human mouth also consider artificial states due to operations of different kinds; to clasps, appliances or dentures of metal or vulcanite. These things cause fermentation and chemical processes of decomposition to an extent which is inconceivable in a normal state of the mouth.

General Summary.

From studies, investigations and experiments published in this work the following results can be compiled.

I. The laws of the development and the systematic of dental caries: —

a) The genetic difference between primary and secondary caries;

b) The genetic difference between natural, non-natural and artificial caries;

c) The genetic difference between crown and root caries;

d) The explanation of latent caries;

e) The explanation of symmetric caries;

f) The geometric laws of (early) caries;

g) The development of caries in the direction of the apex.

II. The existence of the caries canal:

a) being a physiologico-pathological connection between caries and pulp, containing

b) iron and;

c) blood.

III. Determinations of the very peculiar properties of the carious dentine, due to transformation. This transformation tends to form a semipermeable membrane which hinders foreign toxic substances from entering the body.

IV. The osmotic theory as explanation of the initial origin of caries, from which follows: —

a) that natural caries cannot be the result of pure objective factors;

b) but is the result of reciprocal action between the living element of the tooth and certain agents soluble in saliva and able to develop osmotic superpressure to the blood of the pulp;

c) These objective factors are: —

1. Sodium chloride
2. Sugar of different kinds
3. Medical salts
4. Different chemical preparations used as spices condiments etc.

V. Sodium chloride may in overconsumption be regarded as a physiological poison to the human mouth.

General Conclusion.

In this work it is thus fully established that osmosis can take place through the teeth under otherwise certain conditions. This is confirmed by other authors. But this fact does not only involve that osmosis is able to take place through the teeth, but that it *must* take place under certain and commonly occurring conditions. Granted that osmosis takes place, the absolutely inevitable consequence will be that plasm from the blood vessels of the pulp is forced up to the osmotic entrance on the tooth, contibuting to the formation of the carious area, and this quite independently of whether or not the »membrane» is permeable to the salts of the saliva and to those of the blood.

Further we know with certainty that between the carious initial and the pulp exists a clearly visible canal containing blood matter; that in the carious matter bacteria are to be found, successively breaking down the dental tissue; that the microorganisms of caries cannot exist solely upon the dentine and the enamel; and that plasm offers a very good substratum to the microorganisms.

We also know that into the human mouth enormous quantities of osmotically active substances are introduced; that such substances give the saliva a considerable osmotic strength; and that this power, when directed against the teeth, forces the plasm of the pulp to the saliva.

Is any more evidence really needed?

BIBLIOGRAPHY.

Adloff, Walkhoffs Theorie der Zahnkaries etc., D. M. f. Z.[1]) 3. 1914. 169
» » » » » » » » » 6. 1914. 454
» » » » » » » » » 12. 1914. 836

Arkövy, Mikroorganismen der Mundhöhle, Österr.-ungar. Vierteljahrschr. f. Zahnheilkunde 1908.

Auston, Brit. Med. Journal, 1910. 621, ref. Marshall.

Baumgartner, Ueber das Wesen der Zahnkaries etc., D. M. f. Z. 5. 1911. 321

Beck, Dental Caries etc., Dental Cosmos 1918. 351

Berkeley and Hartley, Proceed. Roy. Soc. 73. 1904. 436

v. Beust, Arch. f. Zahnheilk. 10. 1911; 1 and 2. 1912; 11. 1913

Bigelow and Bartell, Journal Amer. Chem. Soc. 29. 1907. 1675, ref. Höber, 1911 p. 190.

Black, An Invest. of the Physical Characters of the Human Teeth, Dental Cosmos 1895

—, Operative Dentistry I 1908

Bluntschli, Zur Frage nach den Ursachen der Karies, Schweiz. Vierteljahrschr. f. Zahnheilk. 3. 1913

Boedecker, Enamel, caries and immunity, Dental Review 5. 1915

Bois-Reymomd, Physiologie des Menschen u. der Säugetiere 1913.

Brasch, Atrophie der Pulpa, D. M. f. Z. 6. 1912. 411

Brugnatelli, Etiologia della Carie dentale 1913

v. Bunge, Zur Ätiologie der Zahnkaries, Schweiz. Vierteljahrschr. f. Zahnheilkunde 1905

Bunting, Comments on an Article by Dr Ragnar Eckermann, ref. Dental Record 10. 1918. 444

Bunting and Rickert, The Tooth a Permeable Membrane, Journal etc. of Nat. Dent. Ass. 5. 1918. 579

Bunting and Wixon, Dental Caries, The Journal of Nat. Dent. Ass. 1. 1917. 18

[1]) D. M. f. Z. Deutsche Monatschrift f. Zahnheilkunde.

Burchard and Inglis, Dental Pathology and Therapeutics 1912
Bünte und Moral, Untersuchungen über den osmot. Druck einiger Lokalanaestetica, D. M. f. Z. 2. 1910. 81
Bönniger, Arch. f. Exp. Pathologie L 76. 1903
Caush, Dental Cosmos 1905
Caush, Dental Record 2. 1915; 8. 1915; 2. 1916
Colyer, Stanley, Dental Caries among certain Central African Races, Dental Record 1 and 12. 1916; 9. 1917
Cohn, Zahnheilkunde 1900
Drucker und Schreiner, Biolog. Centralblatt 33. 1913
Eckermann, Det presumtiva sambandet mellan tändernas och de lösa jordlagrens kalkhalt, Svensk Tandl. Tidskrift 1908
—, Das Eckzahnproblem und die Prämolaren-Wanderung, D. M. f. Z. 12. 1908
—, Physiologische Bedeutung der Kieferbelastung u. Ihre Ermessung, D. M. f. Z. 9. 1911
—, Die Oraldiffusion u. die Ätiologie der Zahnkaries, Svensk Tandl. Tidskrift 4. 1913
—, Aetiology of Dental Caries, Dental Record 12. 1914
—, Karies Etiologi, Svensk Tandl. Tidskrift 1. 1915
—, Fakta i Karies Etiologi, Skand. Tandl. Föreningens Halfsekelsjub. Förhandl. 1916 (Facts in the Etiology of Dental Caries, Dental Items of Interest 6. 1918. Transl.)
—, Articles on »Aetiology of Dental Caries» in Dental Record 1915, 1916, 1917 and 1918
—, »The Tooth a Permeable Membrane», Criticism, Svensk Tandl. Tidskr. 5. 1908. 309
Euler, Das Morgensternsche System etc. D. M. f. Z. 12. 1911. 865
Fano and Botazzio, Arch. de Ital. Biol. 26. 1896. 45
Feiler, Die sogenannte zirkuläre Karies, Deutsche Zahnheilkunde von Witzel 29. 1913
Fischer, Die Biologie der menschl. Zahnpulpa, D. M. f. Z. 1. 1910. 1
—, Atrophie der Pulpa D. M. f. Z. 2. 1910 and 8. 1912.
Fleischmann, Die temporäre Disposition zur Karies, Österr. Zeitschrift f. Stomatologie 5. 1913
Gassmann, Zeitschrift f. Physiolog. Chemie 6. 1908. 455
Gies, Journal of Allied Dent. Soc. 1916
Gottlieb, Exp. Untersuchungen über den Kalkstoffwechsel, Österr. Ung. Vierteljahrschrift 1913. 470
Grieves, Infections resulted from lowered Vitality, Dental Cosmos 11. 1913. 1102
Grüner, Die Beziehungen der Menstruation, der Schwangerschaft

etc. zu Erkrankungen der Zähne, Deutsche Zahnheilkunde 34. 1915

Hamburger, Osmotischer Druck und Ionenlehre, I. 1902. II. und III. 1904

—, Osmotischer Druck 1912

Hammarsten, Lärobok i Fysiologisk kemi 1889

Haus, Speciel Tandanatomi 1917

Head, Dental Cosmos 1907. 802; 1. 1910

Hedin, Pflügers Arch. 60. 1895. 360

Hopwell-Smith, Concerning the Production of Dental Caries, Dental Cosmos 5. 1915

Heidenhain, ref. Hamburger III 1904. p. 169

Hredsky, Zur Ätiologie der Zahnkaries, ref. D. M. f. Z. 5. 1915

Höber, Physikalische Chemie der Zelle u. der Gewebe 1911

Johnston, Hvardagslifvets Kemi, Transl. 1882

Kahlenberg, ref. Hamburger III 1904 p. 169

Kantorowics, Zur Histogenese etc. des Ersatzdentins, D. M. f. Z. 8. 1910. 545.

—, Die Prophylaxe der Zahnkaries D. M. f. Z. 12. 1912. 922

Kantorowics und Speyer, Exp. Untersuchungen über die Beziehungen des Rhodangehalts des Speichels zur Karies, D. M. f. Z. 2. 1914. 81

Keane, Ethnology 1901

Klöser, Ueber keilförmige Defekte, D. M. f. Z. 3. 1914. 216

Koeppe, Bois-Reymonds Arch. 1895. 154

Kunert, Der Wert der modernen Ernährungsreformbestrebungen 5. 1915. 153

Langley and Fletcher, Phil. Transactions Royal. Soc. 1889. 109

Lazarus-Barlow, The Journal of Physiology 19. 1895. 140

Liesegang, Ueber die Zahnentkalkung während der Gravidität, D. M. f. Z. 5. 1915. 187

Levy, Stat. Untersuch. über den Zusammenhang von Zahnkaries, Gicht und Rheumatismus, D. M. f. Z. 6. 1904. 433

Manning, Electrobiolytic Theory of Dental Caries 1. 1919. 21

Magitôt, Recherches sur la carie dentaire 1866

Marshall, Composition of Saliva in Rel. to Dental Caries, The American Journal of Physiology 1915, 1916, 1917

Meyer und Gottlieb, Experimentelle Pharmakologie 1914

Mex, Karies an einem retinierten Zahn einer 68 jähr. Frau, D. M. f. Z. 5. 1914

Meyer, Analyse und Konstitutionsermittlung org. Verbindungen 1917

Michaelis, Wasserstoffionenconzentration 1914

Michel, Die Mundflüssigkeit etc, Deutsche Zahnheilkunde (Witzel) 10. 1909
—, Die konservierende Zahnheilkunde 1912
Miller, Die Mikroorganismen der Mundhöhle 1889. German Edition
Mohr und Stehelin, Handbuch der Inneren Medizin 1912
Molisch, Micro-chemie der Pflanzen 1913
Morgenstern, Basis für die Ernährung in den harten Zahnsubstanzen, Schweiz. Vierteljahrschr. 1901. 185
—, Untersuchungen über die anatom. Grundlagen etc. in den harten Zahnsubstansen, D. M. f. Z. 10. 1909. 713
Mummery, Some Experiments on the Rel. Susceptibility of Different Teeth to Dental Caries, Proceed. of the Royal Soc. of Med. Vol. III. 5
Mühlberger, Grundzüge der pathologisch-histologischen Technik 1912
Neuberg, Der Harn, I and II, 1911
Overton, Pflügers Arch. 92. 1902. 115
Pickerill, The Structure of Enamel, Dental Cosmos 10. 1913. 969
—, Prevention of Dental Caries. The Sixth International Dental Congress London 1914
—, Immunity to Dental Caries, Dental Record 8. 1918. 338
Preiswerk, Beitrag zur Ätiologie der Zahnkaries, D. M. f. Z. 1902
Read, Do Vitamines Exist? Medical times, ref. Dental Record 3. 1919. 117
Riechelmann, Der Entlastungsbügel, D. M. f. Z. 8. 1912
Rygge, Ueber die Schmelzbildung und Schmelzstruktur, Skand. Tandl. Föreningens Halfsekelsjubileum 1916
Römer, Atlas der patologisch-anatomischen Veränderungen der Zahnpulpa 1909
Röse, Die Zähne der Dalarner und Gottländer, D. M. f. Z. 1904. 735
—, Erdsalzarmut und Entartung, D. M. f. Z. 1908
Schaer, Zeitschrift für Analytische Chemie 8. 1903. 42
Scheff, Zahnheilkunde 1902
Squire, Neuralgia, ref. Dental Record 7. 1915. 499
Sundberg, Medicinsk bakteriologi och immunitetslära 1915
Thorsen, Tandens gingivale parti etc., Skand. Tandl. För. Halfsekelsjub. 1916
Thourén, En studie över det normala bettet, Odontologisk Tidskr. 3. 1910. 298
The Dental Art in Ancient Times, Souvenir of the 6th Internat. Dental Congress, London 1914
Tokla, Fra praxis, Norsk Tandläge-forenings Tidende 1. 1918

Trueb, Grössenverhältnisse des Cavum pulpae nach Altersstufen, D. M. f. Z. 6. 1909. 401
Türkheim, Zur Kenntnis der Schmelzhypoplacien, D. M. f. Z. 10. 1914. 729
Walkhoff, Mikrophotograph. Atlas 1897
—, Die Erdsalze in ihrer Bedeutung für die Zahnkaries 1913
Wallace, Sim, The Prevention of Dental Caries 1911
—, Dental Diseases in Rel. to Public Health 1914
Witzel, Deutsche Zahnheilkunde 1913
Äyräpää, Om tandrötans etiologi, Svensk Tandl. Tidskr. 5. 1912